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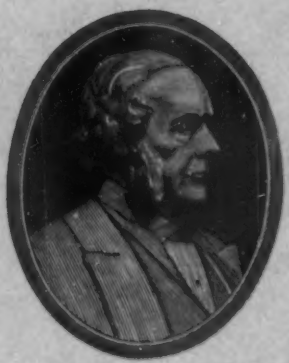

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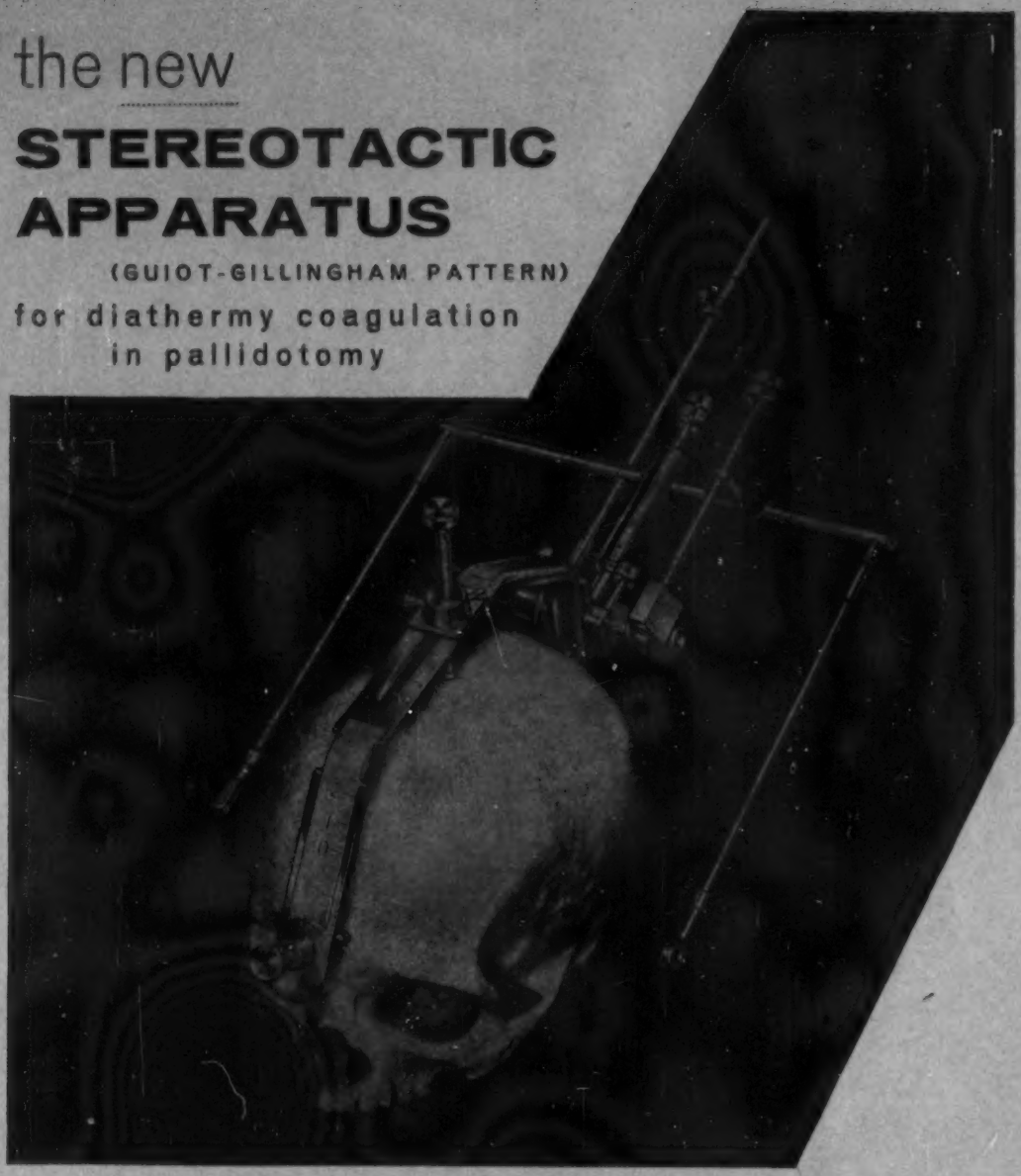

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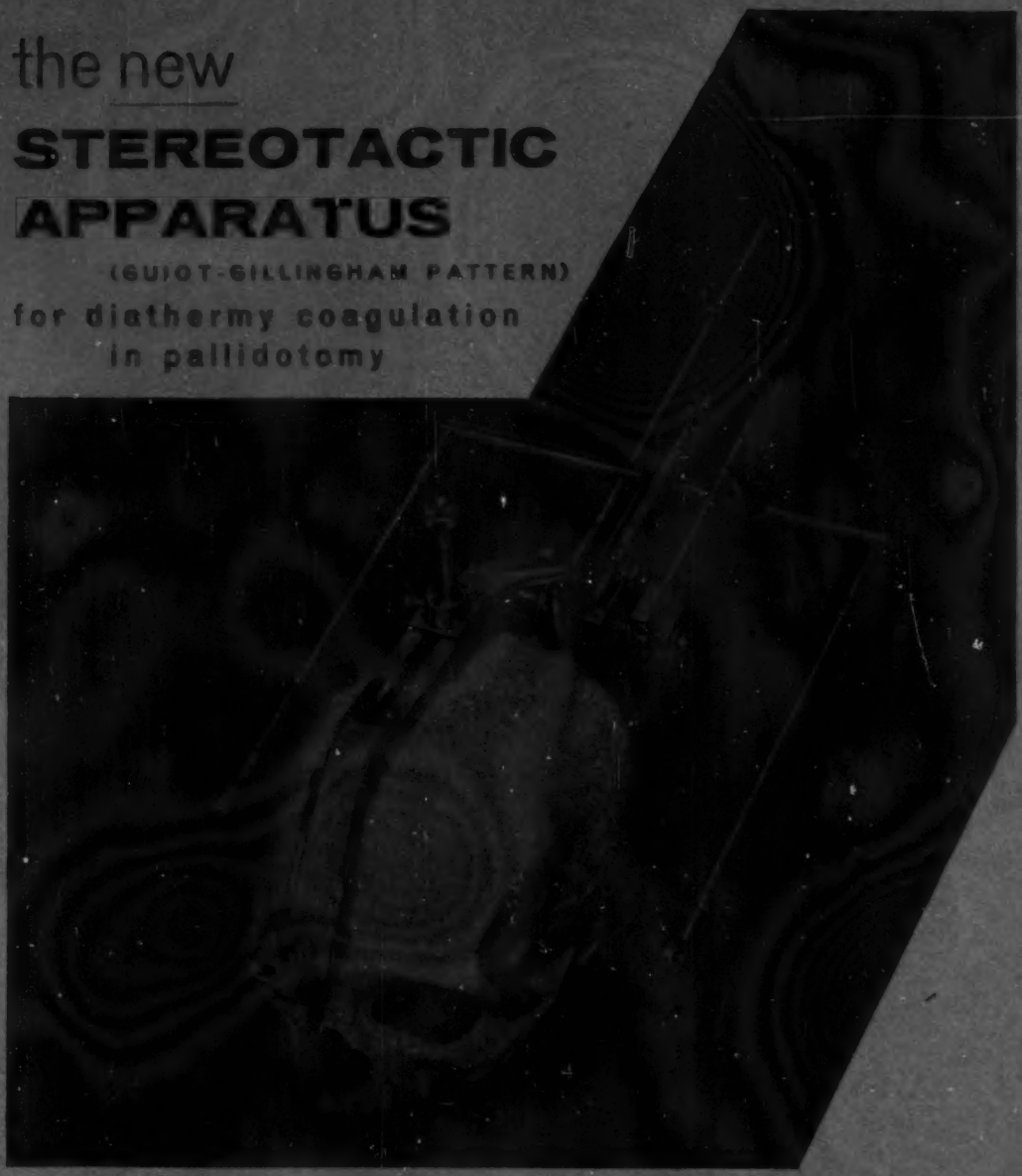

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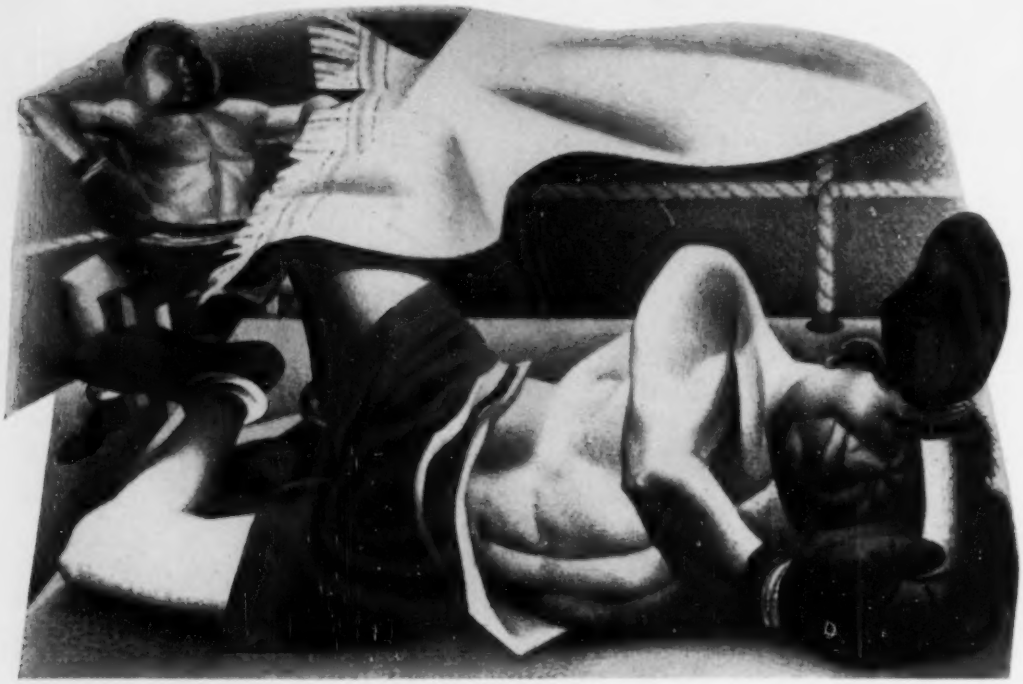

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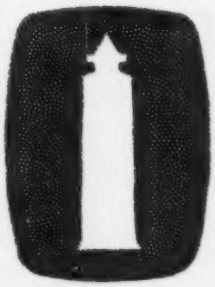

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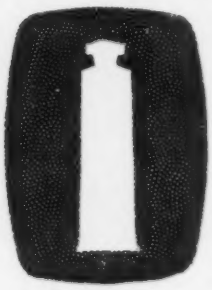

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GLIEDMAN, M., GRANT, R., ROGERS, C., and KARLSON, K. E.: Clorpactin, A Surgical Adjunct; Tumorcidal and Antimicrobial Properties, "Surgical Forum" of the American College of Surgeons, Vol. VIII, 1958.

SOUTHWICK, HARRY W., and COLE, WARREN H.: Prophylactic and Adjuvant Measures in the Treatment of Carcinoma of the Colon; N. Y. State Jour. of Med., P. 86-103, Vol. 59, No. 1, Jan. 1, 1959.

BACON, HARRY E., and BERKLEY, J. L.: Refinements in Prevention of Recurrent Carcinoma Prior to and During Resection of the Colon and Rectum; Preliminary Report; J. of the Int'l. College of Surgeons, Vol. 30, No. 5, Nov. 1958.

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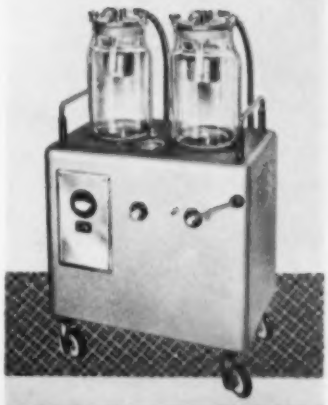

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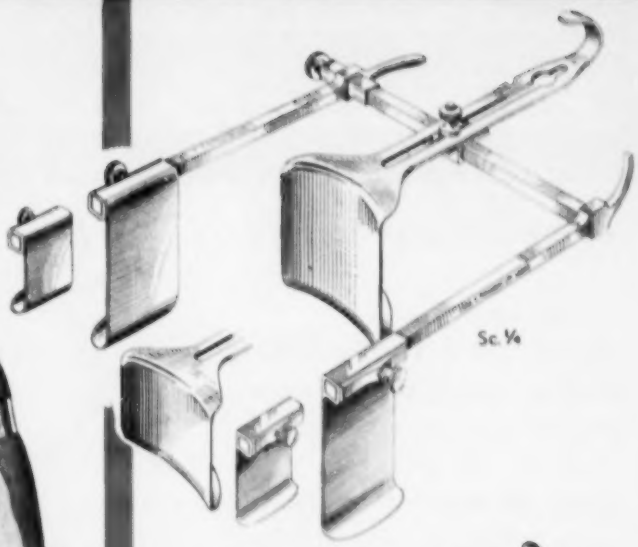

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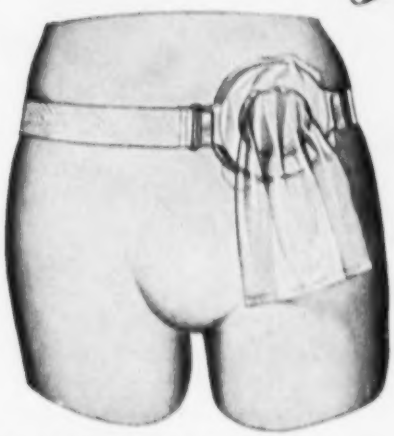

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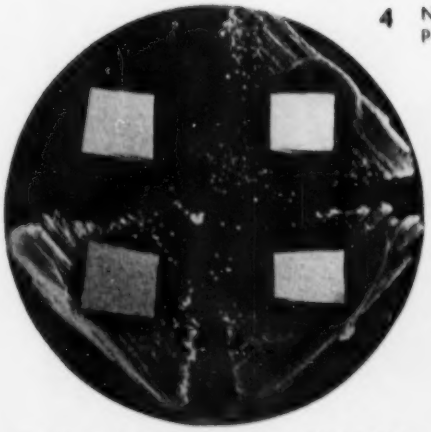

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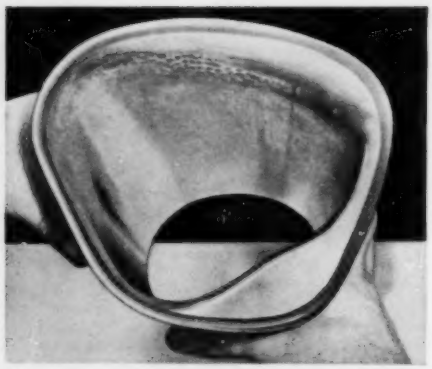

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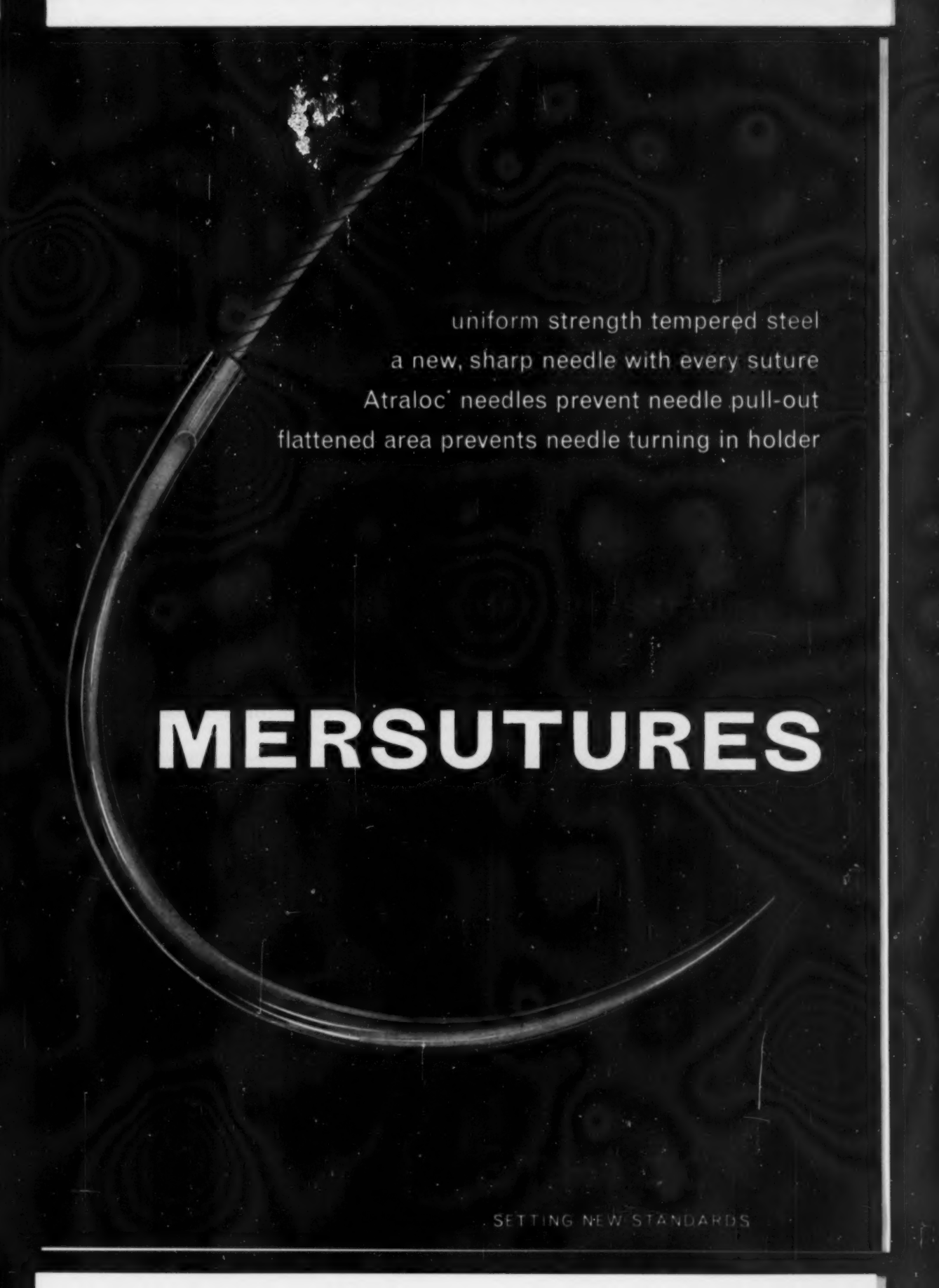
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THE
BRITISH JOURNAL OF SURGERY

VOL. XLVIII SEPTEMBER, 1960 No. 208

VISITS TO CLINICS

THE PROFESSORIAL SURGICAL CLINICAL UNIT AT ST. BARTHOLOMEW'S HOSPITAL

UNDER THE DIRECTION OF PROFESSOR SIR JAMES PATERSON ROSS, K.C.V.O., M.S., F.R.C.S. PRESIDENT OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

"Properly planned and carefully conducted medical education is the essential foundation of a comprehensive health service. The spirit of education must permeate the whole of the health service and that service must be designed and conducted so that, among other things, it secures for medical education the necessary staff, accommodation, equipment and facilities. Medical education cannot be regarded as merely incidental to the hospital service."—Report of the Goodenough Interdepartmental Committee on Medical Schools (1944), *para.* 18.

THE GENESIS AND ESTABLISHMENT OF PROFESSORIAL CLINICAL UNITS

MEDICAL schools had their origin in the informal arrangements made by the medical staffs of hospitals, and later when a university was established in the same city they became the Medical Faculty. The clinical teaching, however, remained an incident in the life of a busy consultant who had received promotion on a basis of seniority, and only attended his out-patient clinic and visited his wards when he had time. The classification and scientific study of clinical material was consequently often deficient. There was not a comparatively permanent and scientific staff, without which continuity and thoroughness of study is impossible. The establishment of a professorial clinical unit is consequently not a sudden outcrop, but is a further step in the long process of evolution. Early in the present century (1908) the Board of Education began giving 'Grants in Aid' to subsidize the medical schools. In 1918 the President of the Board asked Sir George Newman, the Medical Assessor to the Universities' Branch of the Board, to furnish him with a memorandum published as *Some Notes on Medical Education in England.*

In the meantime (1913) the Haldane Commission had been inaugurated to inquire into and report on 'The University Training in London'. The professorial clinical unit system was devised as a result of the criticisms of the traditional system of clinical training of students given in evidence before the commission. These criticisms were strongly supported by the evidence of Abraham Flexner, whose book *Medical Education in Europe* was published in 1912, and also by that of Sir William Osler, the Regius Professor of Medicine at Oxford (1905–19), who had had previous experience in the formation of a clinical unit under a director at the Johns Hopkins Hospital in Baltimore, to which he was appointed as a physician in 1889. This was followed in 1923 by another memorandum by Sir George Newman, addressed to the Minister of Health, on *Recent Advances in Medical Education in England*; both memoranda strongly supported the idea of the establishment of clinical units. The idea was accepted in London by the authorities of the medical school, in consultation with the university, at the following hospitals, University College, St. Thomas's, St. Bartholomew's, and St. Mary's, and in the provinces by the University of Wales at Cardiff (1921), and subsequently in other schools. In the meantime, a deputation from the universities had waited on the Government (June, 1919), as a result of which the Treasury, in consultation with the President of the Board of Education, the Secretary for Scotland, and the Chief Secretary for Ireland, had appointed a University Grants Committee (1920) which has functioned ever since, "to enquire into the financial needs of University Education in the United Kingdom and to advise the Government as to the application of any grants that may be made by Parliament towards meeting them".

Since the second world war changes have occurred in medical education brought about by the recommendations from the Goodenough Interdepartmental Committee on Medical Schools (1944); the implementation of the National Health Act (1948), when the Teaching Hospitals became the property of the State with a relationship between it and the School; and also by the increase of scientific knowledge and its application to medical problems. This increase has resulted in "an enlargement of the outlook in medicine from an art of empiricism and observation to an applied science founded on research; from a craft of tradition to an applied science and law; from a descriptive code of surface phenomena to the discovery

of deeper affinities; and from a set of rules and axioms of quality to measurement of quantity".

The professorial clinical unit must develop these changes. In order to do so there must be a Professor or Director who will devote the whole or the greater part of his time to teaching, treatment, and research.

FIG. 148.—Sir James Paterson Ross talking to one of his patients.

He must above everything else be competent to teach, be of sound scientific training, and possess scientific imagination. He must also have an adequate whole-time or part-time staff, the control of wards containing fifty to one hundred beds, a properly equipped out-patient department in which students can see and learn the early signs of disease, a well-equipped operation theatre, ample laboratory accommodation and equipment for research and pathological work, and ample accommodation for lecturing and the storing of records.

THE CLINICAL UNIT AT ST. BARTHOLOMEW'S HOSPITAL

A professorial surgical clinical unit was established in the Medical College of St. Bartholomew's hospital in 1920 under Professor George Gask (1875–1951), who both before and during the first world war had taken an active interest in the suggested formation of such whole-time units. The success of the unit at Bart's was due to the enthusiasm of George Gask and his team, which included Thomas Dunhill as his deputy, and in which James Paterson Ross was appointed a house-surgeon in 1923. He has remained in the unit ever since, and in 1935 succeeded George Gask when the latter reached the then age for retirement. He succeeded him in a very literal sense. Both were Chairmen for some years of the Editorial Committee of this JOURNAL; both served a full term on the Council of the Royal College of Surgeons of England; both were Hunterian Professors of the Royal College of Surgeons; the main interest of both was the surgery of function, and both found time to write a text-book on surgery.

The personality of the two men seems to have been transmitted from the one to the other. Sir James Paterson Ross can best be described in the words used by the writer who in *The Times* of Jan. 18, 1951, wrote of Professor George Gask: "In spite of leading so full a life for so many years George Gask was scarcely ever seen to show signs of fatigue. He was endowed with a fine physique, and his abstemious and orderly life helped to keep him always in fighting trim. But of even greater value than this was his calm unworrying temperament. He was never ruffled, even in the most trying circumstances. He was an adept in managing his fellow men, and did it without roughness, so that he could always command the loyalty and affection of his colleagues. His surgical technique reflected his mind, being gentle and unhurried. The numerous students who enjoyed his teaching, spiced as it was with quiet humour, carried away with them an invariably affectionate memory."

The surgical clinical unit at St. Bartholomew's Hospital has charge of fifty beds. The unit, like the other surgical firms, takes part in the routine emergency work in the hospital, but, in order that more clinical research may be done, the *ideal* number of beds should be at least seventy.

As the number of students beginning clinical studies each year in St. Bartholomew's is approximately eighty, it is impossible for them all to fulfil a period of surgical dressership working in the surgical clinical unit. The introductory course, therefore, is undertaken, in collaboration, by the Professors who direct the Departments in Medicine, Surgery, and Pathology. Every student is thus afforded the opportunity, during this introductory course before the clinical period, of establishing relations with the professorial clinical units. During this course the students also attend classes in the Department of Physiotherapy, on the sterilization of surgical materials, and are attached to a surgical ward to learn the common nursing procedures, including the possible sources of 'hospital infection.' Each week the unit provides for the instruction of undergraduates working in it by three operation sessions, exclusive of emergency cases, three ward rounds, and one out-patient clinic. There is only one 'waiting list' of patients to be admitted, and so all patients are 'unit patients', but the name of the surgeon who carries out any surgical procedure is on the ward notes which are criticized and corrected by the member of the staff who is in charge of the teaching ward round.

During emergency duty the Director is on duty during the day, and the Deputy Director during the night, to decide on the disposal of cases after admission. Each Monday the Deputy Director and the rest of the staff do a ward round and arrange the week's work, especially with regard to the 'fluid' allocation of patients to particular members of the unit who have a 'research' interest in the clinical condition of the patient. There is always an assistant or registrar on duty to whom a house-surgeon can refer any problem which, if more than routine, is referred to the Director or his deputy. If an assistant or registrar has been following a certain patient in the out-patient clinic, he is given charge of this patient when admitted, and he will probably be asked, when the Director or his deputy make up the list for an operation session, to carry out the surgical procedure.

By this method of sharing out the work the members of the unit team feel that they are more than a mere 'pair of hands'. Anyone who operates on any patient in the unit is responsible for the subsequent treatment under the Director or his deputy.

All members of the unit team join in a staff round every Wednesday morning which lasts at least two hours. All the patients are seen, and each case is presented by the member who has made the preliminary investigations or done the operation. The treatment, and prognosis of thrombosis and embolism. The atmosphere produced by the discussion of such problems as these makes a visit to the staff round of the surgical unit of St. Bartholomew's Hospital a memorable and stimulating occasion.

FIG. 149.—A ward round with Sir James Paterson Ross.

team is always joined by the physiotherapist and often by a radiologist with an interest in diagnosis; there is a special 'follow-up clinic' with a radio-therapist twice each month. On Thursday afternoons there is a routine 'follow-up clinic' for all those who have been in-patients under the unit. There may also be specially arranged sessions for particular cases such as diseases of the breast, thyroid, or vascular system.

Research Programme.—Each member of the unit team is expected to be engaged in a research project, and every encouragement is given by discussion within the unit, by co-operation of those with experience in the basic sciences or in any branch of medicine which may have a bearing on the subject, and by the provision of adequate equipment with laboratory accommodation. The research projects at the present time might be described as investigations of the basic principles which are the controlling factors in the realization of successful surgical procedures, such as investigation of the circulation, both vascular and lymphatic, by arteriography and lymph-angiography; the use of plastic prostheses to replace vascular segments to provide an adequate by-pass for severe ischæmia; and the effects of these on the distal blood-pressure, and from these to obtain some idea of the availability of tissue oxygen for the relief of incipient gangrene. The outcome of ischæmia must depend on the peripheral circulation, and the degree of the latter will determine successful skin-grafting and the healing of a wound. The ideal treatment of a wound must have been the first piece of medical research suggested to primitive man, but it is not yet solved.

To increase the individual resistance to wound infection is also one of the projects being investigated in the unit. No certain method has so far been evolved, but bound up with the answer is the aetiology,

A big factor in the education of the medical student is the uncertainty which is inherent in the subjects. The student hears a different explanation expressed by the teacher in one out-patient clinic

FIG. 150.—Sir James Paterson Ross operating in Theatre G.

from the one he had recently heard concerning the same problem in another clinic. This is educative for the good, thoughtful student, but for the bad one it confirms his opinion that diagnosis is guesswork and fortuitous, as in any other game of chance. To overcome these ideas the Professor, as often as possible, invites a well-known teacher from this or any other country to act as a temporary director of the unit for a period of one to four weeks. The list of those who have so acted in the surgical clinical unit of St. Bartholomew's is imposing, and includes those whose names are known throughout the surgical world.

The Professor.—In October, 1960, Sir James Paterson Ross, having reached the age of automatic retirement, will cease to be the Professor and Director of the Surgical Clinical Unit at St. Bartholomew's Hospital. His services, however, will not be lost to academic medicine, and the authorities of the University of London have appointed him to succeed Sir Francis Fraser as Director of the British Postgraduate Federation from Oct. 1, 1960.

All who know him will join in wishing him many years of health and happiness in his new sphere.

The author gratefully acknowledges the help he has received in the preparation of this article from W. R. Le Fanu, Esq., Librarian of the Royal College of Surgeons of England, and also from the Deputy Director (now the Director) Mr. G. W. Taylor, and other members of the Professorial Surgical Clinical Unit of St. Bartholomew's Hospital.

PUBLICATIONS BY JAMES PATERSON ROSS

1920—"The Influence of Coagulation upon the Reaction of Blood", *J. Physiol.*, **54**, xxx.
1922—"The Reaction of the Blood in Relation to Dyspnœa" (with F. R. Fraser and N. B. Dreyer), *Quart. J. Med.*, **15**, 195.
1924—"A Visit to Certain Surgical Clinics in the United States of America", *St Bart's Hosp. J.*, **31**, 50.
1924—"A Note on Two Cases of Implantation Dermoid Cyst", *St Bart's Hosp. Rep.*, **57**, 148.
1924—"On Reading Surgery", *St Bart's Hosp. J.*, **32**, 10.
1926—"Atrophic Disease of the Shoulder Joint", *Brit. J. Surg.*, **14**, 206.
1926—"The Surgical Anatomy of Head Injuries", *St Bart's Hosp. J.*, **33**, 67.
1927—"Cholecystography", *St Bart's Hosp. Rep.*, **60**, 147.
1927—"The Gradual Relief of Chronic Retention of Urine", *St Bart's Hosp. J.*, **34**, 195.
1928—"Intracranial Injuries", *St Bart's Hosp. Rep.*, **61**, 112.
1929—"An Indication for Ventriculography", *St Bart's Hosp. J.*, **36**, 70.
1929—"Notes on the Barbers' Hall in London", *Arch. Surg., Chicago*, **18**, 1637.
1929—"Cholecystography", *St Bart's Hosp. J.*, **37**, 39.
1930—"Radium and Nervous Tissue" (with E. A. Carmichael), *Proc. R. Soc. Med.*, **23**, 27.
1930—"Richard von Volkmann", *St Bart's Hosp. J.*, **38**, 47.
1930—"Tumours of the Brain and Spinal Cord", Chapter 71 in Bowlby and Andrewes's *Surgical Pathology*, 8th ed. London: Churchill.
1931—"The Anatomy of the Spinothalamic Tract in Relation to Cordotomy", *Proc. R. Soc. Med.*, **24**, 85.
1931—"Anæsthesia before the Modern Period", *St Bart's Hosp. J.*, **38**, 231.
1931—"The Treatment of Cerebral Tumours with Radium", *Brit. J. Surg.*, **18**, 618.
1932—"Causalgia", *St Bart's Hosp. Rep.*, **65**, 103.
1932—"Antonio Scarpa", *St Bart's Hosp. J.*, **40**, 38.
1933—"Sympathectomy for Raynaud's Disease, Causalgia, and Angina Pectoris", *St Bart's Hosp. Rep.*, **66**, 36.
1933—"Sympathectomy as an Experiment in Human Physiology", *Brit. J. Surg.*, **21**, 5.
1934—"Hypodermolithiasis", *Brit. med. J.*, **1**, 235.
1934—"The Prognosis of Raynaud's Disease", *Lancet*, **2**, 97.
1934—*The Surgery of the Sympathetic Nervous System* (with G. E. Gask). London: Baillière, Tindall, & Cox.
1934—"Chondromata of the Long Bones", *St Bart's Hosp. J.*, **41**, 69.
1934—"The Value of Sympathectomy", *Med. Pr.*, **189**, 521.
1934—"The Congenital Short Œsophagus", *St Bart's Hosp. J.*, **41**, 223.
1935—"Cisternal Puncture", *Ibid.*, **42**, 88.
1935—"The Training of a Surgeon", *Proc. R. Soc. Med.*, **28**, 46.
1935—"The Results of Sympathectomy", *Brit. J. Surg.*, **23**, 433.
1935—"The Recognition of Structural Changes in the Arteries in Raynaud's Disease", *St Bart's Hosp. Rep.*, **68**, 121.
1936—"The Surgery of Toxic Goitre", *Nurs. Times.*
1936—"Paget's Disease of the Nipple", *Lancet*, **1**, 1025.
1936—"Advice to a Dresser", *St Bart's Hosp. J.*, **43**, 187.
1936—"Head Injuries", *Ibid.*, **43**, 218.
1936—"1736–1936 (Jean-Louis Petit)", *Ibid.*, **44**, 45.
1937—"A Case of Multiple Myelomatosis" (with G. Discombe and A. H. T. Robb-Smith), *St Bart's Hosp. Rep.*, **70**, 221.
1937—"Honey and Camomile Tea", *St Bart's Hosp. J.*, **44**, 233.
1938—"Prognosis of Infections of the Hand", *Ibid.*, **45**, 269.
1938—"The Surgical Treatment of Cardiac Pain", *Trans. Edinb. Med. Chir. Soc.*, **117**, 75.
1938—"The Treatment of Angina of Effort and of Spasmodic Angina by Thyroidectomy" (with G. Bourne), *St Bart's Hosp. Rep.*, **71**, 61.
1939—"An Investigation into the Effects of Radium upon Carcinoma of the Breast", *Brit. J. Surg.*, **27**, 211.
1940—"Hospital Infection of War Wounds" (with A. A. Miles, H. Schwabacher, A. C. Cunliffe, E. T. C. Spooner, R. S. Pilcher, and J. Wright), *Brit. med. J.*, **2**, 855.
1941—"Carcinoma of the Thyroid Gland growing into the Internal Jugular Vein", *Brit. J. Surg.*, **28**, 634.
1942—"Sepsis in War Wounds of the Limbs", *Brit. med. J.*, **1**, 589.
1946—*Tuberculous Lymphadenitis*. N.A.P.T. Bulletin. London: N.A.P.T.
1946—"Advances in Surgery", *Practitioner*, **157**, 241.
1946—"The Surgery of Arterial Disease and Injury", *Brit. med. J.*, **1**, 1.
1947—"Harvey Cushing, 1869–1939", *St Bart's Hosp. J.*, **51**, 162.
1948—"Plastic Surgery in the Training of a Surgeon", *Brit. J. plast. Surg.*, **1**, 4.
1950—"Your Patient and You", *St Bart's Hosp. J.*, **54**, 50.
1950—"Then and Now", *Middx Hosp. J.*, **50**, 78.
1950—"The Value of Arteriography in the Diagnosis of Peripheral Vascular Disease" (with C. J. Longland), *Practitioner*, **164**, 518.
1951—*British Surgical Practice* (jt. ed. Sir Ernest Rock Carling). London: Butterworth.
1953—"Man's Search for Health", *St Bart's Hosp. J.*, **57**, 44.
1953—"Some Unsolved Problems in the Surgery of the Sympathetic Nervous System", *Ann. R. Coll. Surg. Engl.*, **13**, 356.
1954—"Science and Surgery", *Bull. Amer. Coll. Surg.*, **19**, 19.
1956—"Œdème Bleu" (with I. Macalpine), *Lancet*, **1**, 78.
1956—"Principles of Surgical Technique", *Brit. med. J.*, **1**, 701.
1956—"Nodular Goitre", *J. R. Coll. Surg. Edinb.*, **2**, 81.
1956—"Postoperative Wound Infection" (with R. A. Shooter, G. Ellis, and G. W. Taylor), *Surg. Gynec. Obstet.*, **103**, 257.
1957—"From Trade Guild to Royal College", *St Bart's Hosp. J.*, **61**, 369.
1959—"The Scientific Approach to Surgery", *Brit. med. J.*, **2**, 26.
1959—"The Vascular Complications of Cervical Rib", *Ann. Surg.*, **150**, 340.

THE DIAGNOSIS AND TREATMENT OF POLYPS OF THE RECTUM AND COLON

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ADENOMATOUS polyps of the rectum and colon are relatively common lesions and early recognition and adequate treatment are of practical importance in view of their known tendency to undergo carcinomatous change. In the majority, recognition is easy and treatment is by standard methods. Diagnosis can, however, be unexpectedly difficult, and the right course of treatment is not always clear, especially when malignant change in the polyp is suspected or proven.

Material.—This paper is based on the clinical and pathological aspects of 30 consecutive patients with polyps seen in the Rectal Clinic of the Middlesex Hospital during 33 months in the years 1955 to 1957.

Of the 30 patients, 17 were men and 13 were women. Polyps are not uncommon in children, but no example was seen in this series. The youngest patient was 27 years old and the oldest 83 years; 13 were aged between 60 and 69 years and 10 between 40 and 60 years.

Of the 30 patients 8 had carcinomatous change in a polyp, and in most there was no distinctive clinical suggestion of this change.

No patient with familial polyposis or inflammatory polyp formation has been included.

Although the total is small, a number of diagnostic and therapeutic problems were encountered and I shall try to show how these were overcome and, at the same time, survey the management of patients with polyps of the large bowel.

DIAGNOSIS

Symptoms.—The great majority of patients presented with rectal bleeding (24 out of 30). Of these patients 9 observed that the blood was dark red in colour, and this often signifies an important lesion above the anorectal region; 6 patients observed that the blood was streaked on the motion, sometimes mixed with mucus; 4 of the 24 patients noted diarrhœa and 2 were constipated; 3 patients reported continued bleeding after the injection of hæmorrhoids.

In the 6 patients who did not complain of bleeding, one had constipation, one diarrhœa, one a sense of incomplete evacuation, one a mucoid rectal discharge, and two were found to have a polyp in the course of examination for other complaints.

Signs.—Abdominal examination was negative in all the patients in this series.

Digital examination of the rectum produced positive information in 8 patients. In 2 there was obvious blood on the finger cot. In 3 a mobile stalked polyp was felt and in these patients it was subsequently possible to deliver the polyp through the anal canal and excise it. In the remaining 3, the lower part of a polypoid tumour could be felt, and in 1 of these palpation under anæsthesia subsequently played an important part in the selection of treatment.

Case 1.—Mr. W. S., aged 53 years, complained of passing mucus per rectum for two years. He had seen no blood. A firm, spherical mass was felt at the finger-tip and a sessile polyp was seen at 14 cm. A biopsy was reported as an 'adenomatous polyp'. In view of this, the patient was examined under anæsthesia, and bimanual

FIG. 151.—*Case* 1. Opened operative specimen, showing sessile polyp of upper rectum. There is a carcinomatous ulcer in the upper edge of the tumour.

palpation showed a solid tumour 4 cm. in diameter. In view of this solid feeling, a radical resection of the polyp was done, with restorative anastomosis. The patient was very fit 2½ years later.

The specimen (*Fig.* 151) showed a sessile, adenomatous polyp, 5 cm. in diameter, with an ulcer in its upper margin; this proved to be an adenocarcinoma of average grade malignancy. Twelve mesenteric nodes were free of tumour.

Sigmoidoscopy is the most informative examination. It was done in 22 out-patients and in 20 useful information was obtained on the first visit. In 9 a polyp was seen. In 10 altered blood was seen on the mucosa, making it likely that a neoplasm would be found higher in the colon. In another patient, fæces, removed through the sigmoidoscope, gave a strongly positive occult-blood reaction.

Immediate out-patient sigmoidoscopy is strongly recommended. In many patients a polyp will be seen and admission arranged at this first visit. In others, the presence of blood on the mucosa will indicate the need for careful investigation. In obtaining this sign it is essential to examine the unprepared bowel, for a rectal washout is very likely to remove this important evidence. In every patient in this series in whom this sign was observed, a neoplasm was subsequently found. Occasionally, this sign is due to free hæmorrhoidal bleeding, but if altered blood is repeatedly seen

this explanation must not be entertained. Hæmorrhoids should be injected at the first attendance so that they cannot continue to bleed.

For out-patient use, a ½-in. diameter sigmoidoscope of Lloyd-Davies or Morgan-Officer type is recommended. This is small enough to cause little discomfort, and to pass easily around lumps of fæces while allowing an excellent view of the bowel.

Whenever a negative examination has been made on a patient with a suspicious history it is worth examining a sample of fæces from the rectosigmoid region for occult blood. Taken at this level, the fæces are unlikely to be contaminated by hæmorrhoidal bleeding.

FIG. 152.—*Case* 2. Barium enema film, showing filling defect at the junction of sigmoid and iliac colon.

Case 2.—Mr. J. S., aged 60 years, had an injection of a hæmorrhoid and 16 months later complained of a little further bleeding. Sigmoidoscopy was negative, but fæces removed through the instrument at 15 cm. gave a strongly positive occult-blood reaction. A barium enema was ordered and showed a doubtful colonic filling defect. Sigmoidoscopy was again negative, but a further barium enema gave a clear picture of a sigmoid polyp, which was removed by colotomy and polypectomy (*Figs.* 152, 153). Recovery was uneventful and microscopy showed a benign adenoma.

When a polyp has been seen, or suspicion aroused by finding altered blood in the bowel, the patient should be admitted for sigmoidoscopy under anæsthesia. For this it is necessary to wash out the bowel so that the sigmoidoscope can be passed as far as possible and a clear view obtained. In this way, 6 more polyps were found.

When a polyp is seen its characteristics should be noted. It may be stalked or sessile. A polyp with a stalk is usually 1–2 cm. in diameter, rounded and smooth, and a brighter red than the surrounding mucosa. A sloughy, septic patch may indicate early carcinomatous change. It is important to see the stalk and assess its length; it is not usually more than 1 cm. wide and consists of normal mucosa and submucosa, drawn out by the weight of the polyp. As will be seen later, the length and thickness of the stalk is of practical importance. Any suggestion of infiltration of the stalk must be noted and this assessment may be helped by moving the polyp with the end of the sigmoidoscope.

Sometimes a villous papilloma will be seen. This is a specific neoplasm which is soft and sessile and covered by innumerable fronds. It may extend widely and is more common in the rectum than in the colon.

The Barium Enema Examination plays an important part in the diagnosis of colonic polyps.

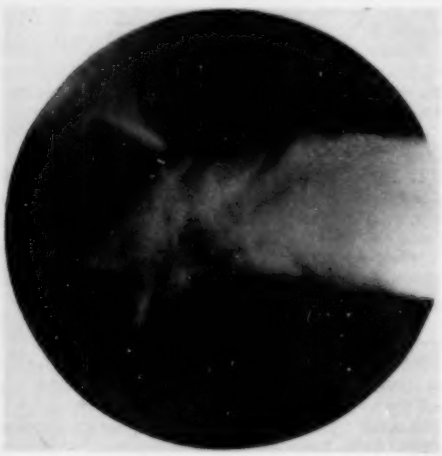


FIG. 153.—*Case* 2. Magnified view of another barium enema film, showing the polyp and stalk in negative shadow.

Direct inspection through the sigmoidoscope should always be used for the diagnosis of rectal polyps, but often the instrument will not pass the rectosigmoid junction. A barium enema must, then, be used for the diagnosis of a colonic polyp. It should be a rule that a barium enema is not ordered before a sigmoidoscopy has been done, and the information obtained should be given to the radiologist.

In this series 20 patients had a barium enema examination and in 13 of them it was the essential investigation leading to a diagnosis of a colonic polyp. The presence of a polyp is suggested by a consistent filling defect, seen in several films taken at different stages of filling and emptying, without that deformity of the bowel which occurs with a carcinoma.

It can be remarkably difficult to demonstrate a polyp in the colon even when its presence is suspected. The negative shadow can be lost in too much barium, or through overlapping of loops of colon. On the other hand, fæces can give a negative shadow that resembles a polyp, so the bowel must be carefully prepared. A filling defect seen in only one film is more likely to be due to fæces than to a polyp.

Among the 13 patients in whom barium enema gave evidence of a polyp, 7 had to have the examination

repeated. One patient had 3, and one 4, enemas before clear evidence was obtained.

Case 3.—Mrs. N. H., aged 62 years, complained of rectal bleeding for 5 months. Sigmoidoscopy showed blood on the mucosa, as it did throughout her investigation. Two barium enemas were done and nothing was found, although she continued to bleed. She was then admitted with a cerebral thrombosis, and, as she continued bleeding, a third enema was carried out, but this was also negative. She was not fit for operation, but

FIG. 154.—*Case* 4. Film from third barium enema examination, showing filling defect in sigmoid colon (arrowed). This film shows the tortuosity of the sigmoid colon, and explains why this polyp was not seen through the sigmoidoscope.

when she returned 5 months later with undiminished bleeding her general condition had improved. In the hope of localizing the tumour a fourth barium enema was done and this showed a polyp in the sigmoid colon. A hard polyp with a long stalk was found and the stalk ligated and divided. She remained well until a further cerebral thrombosis 2 years later. The polyp was found to be invaded by a carcinoma of low-grade malignancy, but the stalk was free of tumour.

Comment.—This lady would have had a laparotomy much sooner but for her general infirmity. The finding of blood on the mucosa at every sigmoidoscopy, with negative barium enemas, made it virtually certain that she had a colonic polyp.

The history of the patient who required three barium enemas is also instructive.

Case 4.—Mr. W. F., aged 77 years, complained of abdominal discomfort and the passage of brown fluid per rectum. At sigmoidoscopy free blood was seen and in the barium enema there was a possible filling defect in the sigmoid colon. A second enema, done with special care, showed no abnormality. It was decided to allow the patient to go home, but as a precaution a sigmoidoscope was first passed. As altered blood was seen all the way to 25 cm. his discharge was cancelled. The case was discussed with the radiologist and in a third enema a definite sigmoid polyp was seen (*Fig.* 154). This was removed by colotomy and proved to be benign.

These case-histories show that the whole clinical picture must be considered in these patients. In particular, the physical sign of altered blood must not be set aside until satisfactorily explained. In a difficult case, the radiologist should be helped by having all the clinical data at his disposal. He should be assisted by a thorough cleansing of the bowel, and here veripaque washouts can be very effective. The use of air insufflation after evacuation of the major part of the barium sometimes gives an excellent picture of a polyp coated with a thin film of barium.

The surgeon should scrutinize barium enema films personally. On two occasions in this series negative reports were given by trainees, and later inspection of the films showed that filling defects were present.

Coloscopy sometimes reveals unsuspected polyps and will be described later.

TREATMENT

The choice of the correct treatment for a polyp depends on its site, size, and pathological character. Broadly speaking, those polyps which are easily felt with the finger can be removed through the anal canal, and stalked polyps seen with the sigmoidoscope can be removed through it. On the other hand, those polyps which can only be found with a barium enema will have to be removed at laparotomy. These broad principles may be modified if the tumour is large or suspicion of malignancy exists. The selection of the correct treatment, and its technique, will now be considered under three main headings:—

1. Polyps which can be removed through the Anal canal.—These are stalked polyps which can be made to prolapse through the anal canal or can be removed through the sigmoidoscope.

a. DELIVERY OF POLYP PER ANUM, WITH LIGATION OF STALK.—This, the simplest method of treatment, has only a limited application, as relatively few stalked polyps occur in the lower rectum, but it is always worthy of trial when the whole polyp can easily be felt with the finger.

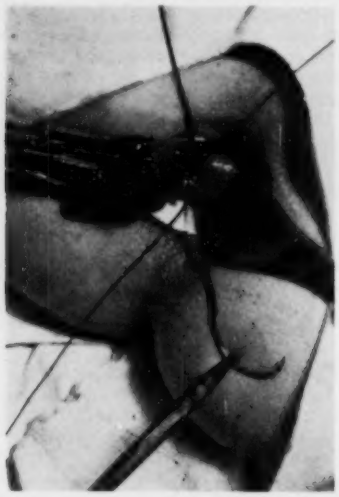
General anæsthesia, with good relaxation, is induced, the anal canal dilated, and the polyp delivered. The stalk is secured by simple ligation or, if the pedicle be broad, by the application of Goodsall's ligature (*Fig.* 155). This ligature has the advantage of leaving no stricture.

Three patients in this series were treated by this method, and one of the cases requires detailed description.

Case 5.—Miss N. F., aged 67 years, gave a history of "something dropping out of the back passage" during defæcation. She had noticed this for 3 months and it was usually accompanied by rectal bleeding. Her bowels moved regularly. On digital examination the ampulla of the rectum seemed to be filled with papillary tumour which felt indurated. At this examination she was thought to have a carcinoma of the rectum. Biopsies were taken from the stalk and fundus of the tumour.

Both biopsies showed parts of a benign polyp, so she was examined under anæsthesia. The rectal ampulla was filled with a papillary tumour, 8 cm. in diameter, which was attached to the anterior rectal wall by a vertical stalk which began 6 cm. from the anal margin. Much of

the tumour was indurated, but the stalk and the surrounding mucosa were supple. After dilatation of the anus it proved possible to deliver the tumour. It was wrapped in a dry swab and firm traction applied. This displayed the stalk, and palpation of it, with one finger in the vagina, showed no induration of the base. It was



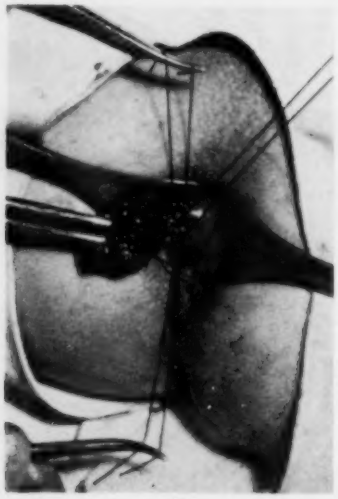
A

C

felt that there was a prospect of local removal, so six Goodsall sutures were inserted in the stalk and tied and the tumour was removed. The patient made an uneventful recovery.

Examination of the specimen showed that some of the muscular wall of the rectum had been drawn into the stalk. The tumour was a papillary adenoma with many areas of adenocarcinomatous change of low-grade malignancy. These had begun to invade the stalk, but they did not extend to the line of division.

The patient attended for examination 4 months later. There was no sign of papillary tumour in the rectum. There was an extensive scar on the anterior rectal wall, but a 1-in. sigmoidoscope passed easily and a polyp at 14 cm. was removed with the diathermy loop. When seen 2½ years after operation the rectum was clear, there was no induration in the mesorectum, and sigmoidoscopy was negative.



B

FIG. 155.—Goodsall's ligature. A, The polyp has been delivered through the anal canal. The first Goodsall suture has been passed and the first loop made. The second Goodsall suture is being passed: the thread will be cut at the needle to make the second and third loops. B, The thread has been cut and the three loops are held in forceps, ready for tying. C, The loops have been tied and the stalk divided. The threads are held until hæmostasis has been checked.

Comment.—This procedure may seem to fall short of that necessary for the adequate removal of carcinomatous tissue. There were two reasons for choosing this method:—

1. Palpation revealed a soft stalk and suggested that the line of division of the stalk would be well clear of any malignant infiltration.

2. The site of removal lay within easy reach of the finger and allowed a careful digital check on the area of removal, and also the mesorectum where any lymph-node recurrence could be felt.

If this tumour had been at a higher level in the rectum this type of follow-up examination would not have been possible. A radical resection would have been indicated and, at this level, a restorative anastomosis would have been possible. If radical excision had been carried out in the patient just described, an excision of the rectum with colostomy would probably have been necessary, so it was felt right to give the less radical method a trial, and to maintain close supervision. This would not have been acceptable if the tumour had been of a high grade of malignancy.

b. SIGMOIDOSCOPIC REMOVAL WITH DIATHERMY LOOP.—This is the method most often used (13 out of 30 patients in this series).

Apparatus.—Most sigmoidoscopes can be used, but it is a great advantage to have proximal illumination. A bulb held distally on a stalk may catch on the snare or be obscured by blood. It is best to use a 1-in. diameter instrument as this gives room in which to manœuvre the loop. Short, wide-bore instruments have been designed by Lloyd-Davies, with a suction

tube, which are valuable for piecemeal treatment of villous papillomata. The diathermy apparatus needed is a Frankfeldt snare and a diathermy button on a stalk.

Technique.—

i. *Pedunculated polyps*: When a polyp is examined it is important to note whether it is stalked or sessile. If it has a thin, supple stalk then the diathermy loop can be used. It is better to admit the patient and give a general anæsthetic.

The left lateral position is generally suitable. A 1-in. diameter sigmoidoscope is passed, the polyp brought into view, and the distance of the stalk from the anal margin noted accurately for use at follow-up examination. The Frankfeldt snare is passed, with the arms of the loop separated by about ¾ in., slipped over the polyp, and eased down the stalk so that it can be tightened on the lower third, as shown in *Fig.* 156.

The diathermy machine is set at a *low* cutting current. It must be emphasized that division of the stalk is achieved much more by the cutting action of the tightened wire loop, and by pulling the polyp into the sigmoidoscope, than by the prolonged application of the diathermy current. Short touches on the diathermy pedal lasting about 0·5 sec. are given two or three times during the closure of the snare. When the wire has cut through, the stump is inspected and if there is any bleeding a swab soaked in 1–1000 adrenaline is applied for a few minutes.

rectum covered by peritoneum. The writer has seen a perforation of the anterior rectal wall 1·5 cm. in diameter 48 hours after the removal of a polyp with diathermy and he has heard of another case. (Neither occurred in this series.) There is no doubt that care had been exercised in these cases and it is clearly easy to overdo the application of diathermy. It may take 15 sec. for the loop to cut through the stalk and diathermy may only be applied for a fraction of this

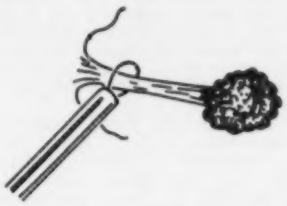


FIG. 156.—Sketch to show the correct point of application of the diathermy snare to the stalk of a polyp.

time. It is also worth remembering that strong traction will pull the muscular wall of the bowel into the stalk, where it can be damaged by the loop. Traction should not be applied while the loop is being eased into place.

The highest point at which a polyp was removed with diathermy in this series was at 23 cm. from the

FIG. 157.—*Case* 6. Barium enema film showing a filling defect due to a polyp in the iliac colon. This was seen after passing the sigmoidoscope to 23 cm.

FIG. 158.—*Case* 6. Straight radiograph of the abdomen. The sigmoidoscope has been passed to 25 cm. and a silver clip applied to the mucosa at this point.

The polyp is laid out and a thread passed through the stalk close to the point of section. If the thread is used to suspend the polyp in formol-saline the stalk is kept straight for accurate histological examination. Alternatively, the polyp can be pinned out on cork.

Use of the diathermy must be particularly restrained when polyps are treated in the colon and in anus, and the history of this patient has several features of interest.

Case 6.—Mrs. B. S., aged 67 years, first complained of rectal bleeding in December, 1954, when hæmorrhoids were twice injected. In February, 1955, she still had rectal bleeding. Blood-clot was found in the rectum and a polyp at 15 cm. was removed. A year later she reported

passing blood-flecked stools, but sigmoidoscopy was negative. A barium enema showed a doubtful filling defect. A further sigmoidoscopy was negative, so she was admitted, prepared with veripaque, and a second barium enema done. This showed a filling defect in the sigmoid colon (*Fig.* 157). It did not look as if a sigmoidoscope was likely to reach this polyp.

At examination under anæsthesia the 1-in. diameter sigmoidoscope would not pass beyond 15 cm., but the ½-in. diameter instrument passed to its full length of 25 cm. and the polyp was seen. The snare was worked around the stalk and a polyp, 1·5 cm. in diameter, was removed. None of the stalk was found on the specimen, so the 1-in. instrument was passed, this time successfully, and the stalk seen at 23 cm. The loop was passed around it and the stalk removed. The polyp was found to be benign.

Surprise was expressed that a polyp which seemed, on X-ray examination, to lie so high could be removed through the sigmoidoscope. When the patient returned 3 months later a 25-cm. sigmoidoscope was passed. No abnormality was seen. A silver clip was placed on the mucosa at the highest point reached and a radiograph taken later, confirming that a sigmoidoscope can occasionally be passed to the iliac colon (*Fig.* 158).

A year later the patient had further bleeding and a small polyp was removed from the splenic flexure by colotomy. A year after this she was well and free from symptoms.

Comment.—1. Some patients form more than one polyp and they should all be warned to return if symptoms recur. It is not possible, nor does it seem to be necessary, to follow up all patients who have had polyps removed, as is done in the case of bladder papillomata.

2. No trace of the stalk of the polyps at 15 cm. and 23 cm. was found at follow-up examination. This is usual if the stalk has been correctly divided close to the bowel wall.

It is important that the thickness of the stalk of a polyp should be assessed when a choice of treatment is being made. The majority of polyps have a thin stalk, but some of the larger pedunculated polyps have a wide stalk containing vessels of some size.

Case 7.—Mrs. J. S., aged 64 years, was admitted with a polyp at 15 cm. It appeared to be about 3 cm. in diameter and to have a short, thick stalk. It was decided to remove it at laparotomy. A biopsy showed no evidence of malignancy so the rectosigmoid junction was mobilized and opened. There was a soft polyp 3 cm. in diameter with a short stalk 2 cm. wide. A Goodsall ligature was inserted and the polyp removed. The ligature embracing the artery of supply was not tied sufficiently tightly and free and forceful bleeding occurred. It was not difficult, with this approach, to secure it and tie it off. Recovery was uneventful.

Comment.—If this stalk had been divided through the sigmoidoscope with the diathermy snare, troublesome bleeding might have occurred and proved difficult to stop.

ii. *Sessile polyps*: These are commonly seen in the course of sigmoidoscopy. Often they are not more than 0·5 cm. in diameter, pale in colour, and prove to be a nodule of hyperplastic mucosa. If the polyp is a deeper red than the surrounding mucosa this suggests that it is an active adenoma. These polyps often cause no symptoms. They can usually be removed entire with the biopsy forceps and should always be submitted to histological examination. When larger, they can be treated by biopsy and coagulation with the diathermy button. When beyond the scope of this treatment, management depends on the site of the polyp. In the lower rectum a combination of the diathermy snare and the button will usually suffice. In the upper rectum this method must be applied with caution and colotomy, or resection, may be a more suitable line of treatment.

iii. *Villous papillomata*: If neglected, these tumours tend to encircle the bowel and produce symptoms by their size and excessive production of mucus. There is a tendency to malignant change and in 10 per cent of rectal carcinomata are said to arise in them. Any indurated area in a villous papilloma is likely to be carcinomatous and a biopsy should be taken. If positive, radical excision will be needed as these tumours are sessile and infiltration of the bowel wall is likely.

By reason of their preference for the rectum they are usually felt on digital examination, and in these circumstances removal through a wide-bore sigmoidoscope can be tried when the whole of the tumour can easily be seen. Piecemeal removal with the snare and gentle coagulation with the button are used. This line of treatment should not be lightly undertaken, especially if the bowel affected is covered by peritoneum. Anterior resection can be done for tumours in the upper rectum (*Case* 12). In the lower part of the rectum considerable efforts would be made to achieve a local removal and, if resection seemed unavoidable, abdomino-anal anastomosis would be very seriously considered.

2. Polyps which can only be removed by Laparotomy.—Polyps which cannot be removed through the anal canal must be approached through the abdomen and, again, two methods of treatment are available:—

a. COLOTOMY AND POLYPECTOMY.—Of the 30 patients in this series 8 were treated by laparotomy, colotomy, and ligation and division of the stalk of the polyp.

Technique.—The bowel is thoroughly cleansed with pre-operative washouts and phthalylsulphathiazole is given. The incision used depends on the position of the polyp, but is most often a left paramedian.

When the affected loop of bowel is delivered the polyp will be detected by drawing the bowel through the fingers. When found, it should be manipulated as far up and as far down the bowel as possible. If the bowel is opened midway between these points, the point of attachment of the stalk should be found. The polyp is withdrawn and the stalk examined. If it consists of normal mucosa it can be ligated and divided, using Goodsall's sutures where necessary.

When the polyp has been removed, it is easy to introduce a sterile sigmoidoscope through the incision, the bellows being sheathed in a tubular sterile towel. Deddish and Hertz (1957) give a detailed description. By this means unsuspected polyps can be found.

Case 8.—Mr. E. P., aged 61 years, had passed clots of blood with the stool for 5 weeks. Sigmoidoscopy showed blood and mucus in the upper rectum, but a barium enema was reported negative. After admission sigmoidoscopy again revealed blood in the rectum. The radiographs were reviewed and a filling defect was seen (*Fig.* 159). At laparotomy, a polyp was removed from the sigmoid colon. A sigmoidoscope was passed and a further polyp seen in the lower sigmoid. This was drawn up through the colotomy wound and removed. Both polyps were benign and the patient made a good recovery.

It is of considerable interest that 3 of the 8 polyps removed by colotomy showed carcinomatous change in an adenomatous polyp. In all 3 cases the stalk was free of invasion and the carcinoma was well differentiated. One patient was 83 years old, one 66 and arteriosclerotic, and one 62 with a recent hemiplegia.

FIG. 159.—*Case* 8. Barium enema film showing a filling defect in the sigmoid colon. None of the films suggested the presence of a second polyp.

In these circumstances colotomy and polypectomy were almost certainly the correct treatment, but consideration of these figures raises the question of the continued use of a local operation such as colotomy and polypectomy.

The position may be summarized in this way: If a polyp is found beyond the range of sigmoidoscopic removal, then a laparotomy must be undertaken for its removal. Either colotomy and polypectomy or resection and anastomosis should be done. The operative risk of colotomy is less than that of resection. On the other hand, if colotomy is preferred and the polyp is later shown to be carcinomatous, the surgeon will be left wishing that he had done the more radical procedure. This dilemma cannot, unfortunately, be solved by the examination of frozen sections at operation, as pathologists do not feel able to give an opinion on this material. Palpation of the polyp and stalk may help, as hard nodules were felt in all 3 patients showing carcinomatous change.

The correct course will vary with the condition of the patient and the operative findings. Provided the bowel is well prepared and not excessively laden with fat, and if a precise operation is carried out by a modern technique (Lloyd-Davies, Morgan, and Goligher, 1953), the risk of resection is probably little greater than that of colotomy. It is suggested that a resection should be done in young patients and in those in whom examination suggests the presence of carcinomatous change. In the aged, who may have an impaired mesenteric circulation, and in those unfit for the bigger procedure, polypectomy may be chosen in the knowledge that it will generally be adequate.

Not all polyps of the colon are tumours of the mucous membrane. The polyp removed from one patient in this series proved to be a simple submucous

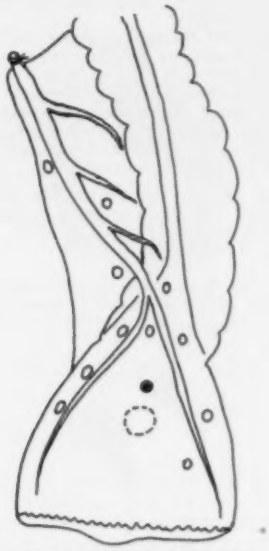


FIG. 160.—*Case* 9. Diagram of the posterior aspect of the specimen obtained by abdominoperineal excision of the rectum. The dotted circle shows the site of the polyp. The solid black circle shows the position of the invaded lymph-node. Open circles represent lymph-nodes found to be free of metastasis.

leiomyoma. Other submucous tumours, such as lipomata, may become polypoid and can precipitate an intussusception.

b. RESECTION OF COLON OR RECTUM.—Resection of the rectum or part of the colon was carried out in 6 patients. In 3 this was done because of evidence or suspicion of carcinomatous change in a polyp, in 2 because multiple polyps were found at laparotomy, and in 1 patient the resection was done for an extensive villous papilloma. The other indication for resection is the discovery of a sessile polyp at colotomy. The shorter the stalk and the harder the polyp, the more desirable will be resection.

Among the 3 patients who required resection of a malignant polyp, one has already been described (*Case* 1). The next patient also illustrates some important principles.

Case 9.—Mr. C. C., aged 73 years, complained of a feeling of incomplete emptying of the rectum for one year. Digital examination revealed a sessile polypoid mass on the right of the rectal ampulla; there was no evidence of invasion of the rectal wall. Material for biopsy was taken from the stalk and the fundus of the polyp. The report stated: "Sections from the superficial part show a simple papilloma, with carcinomatous change in the highest part. One of the pieces from the base is invaded by carcinoma of relatively high malignancy." In view of the latter part of the report, an abdominoperineal excision of the rectum was carried out. The patient was well and free from recurrence 3 years later.

The specimen proved to be of great interest. There was an ulcer at the site of the removal of the biopsy specimens; microscopy of this area showed no evidence of residual growth, although an area of mucoid material was found in muscle deep to the ulcer. Thirteen lymph-nodes were recovered from the mesentery and one, in close proximity to the tumour, was replaced by mucus-secreting columnar-cell carcinoma of average grade malignancy (*Fig.* 160).

Comment.—Although the tumour occupied a similar site, this patient differed fundamentally from *Case* 5 in that biopsy of the stalk showed it to be infiltrated by carcinoma. Invasion of the wall of the rectum had, therefore, to be assumed and a radical excision carried out. This course was fully justified by the finding of a metastasis in a lymph-node. This had occurred before significant infiltration of the bowel wall.

The next patient shows that the application of this knowledge will sometimes, necessarily, lead to the performance of a resection not pathologically necessary.

Case 10.—Mr. L. P., aged 63 years, gave a history of rectal bleeding for one year, the motions being streaked with dark-red blood. Sigmoidoscopy revealed a polyp

FIG. 161.—*Case* 10. Diagram of the polyp showing the extent of the two areas of carcinomatous change.

2 cm. in diameter at 15 cm. Biopsy showed a benign papillary polyp. Sigmoidoscopy under anæsthesia confirmed the presence of a cherry-red polyp with a pale, supple stalk 2 cm. long. This was divided close to the bowel wall with a diathermy loop. Histological examination showed two areas in which the stroma was invaded by carcinoma of average-grade malignancy, although the stalk was free (*Fig.* 161).

For the reasons given later it was decided to resect the affected segment of colon. The patient was placed in lithotomy-Trendelenburg position and a sigmoidoscope passed to 15 cm. The abdominal operator then passed a stitch through the bowel wall opposite the tip of the sigmoidoscope. The colon and rectum were mobilized and divided 15 cm. above and below the marker stitch, and the inferior mesenteric artery divided below the left colic artery. A restorative anastomosis was done and the patient made an uneventful recovery. Histological examination revealed no trace of carcinoma in the bowel or lymph-nodes and the patient was well 18 months later.

Comment.—This patient had a malignant neoplasm invading the stroma of the polyp. If lymphatic metastasis had already occurred it would spread widely before becoming clinically obvious. This type of case will always present a difficult choice and the known risks of resection will have to be set against the possible risk of metastasis.

One patient had a resection for a villous papilloma.

Case 11.—Mr. W. G., aged 68 years, who resided overseas, complained of intermittent rectal bleeding for two years. Sometimes there was also a clear mucous discharge. The bowels were regular. On rectal examination a soft mass was felt at the finger-tip and sigmoidoscopy showed a sessile villous papilloma extending 11–14 cm. from the anus. A barium enema showed many diverticula in the sigmoid, and at laparotomy considerable diverticulitis was found. Excision of the upper half of rectum and the sigmoid colon was performed and an anastomosis done. The patient was in good health one year later. The specimen showed a villous papilloma 4 cm. in diameter which half-encircled the upper rectum.

Comment.—Local removal of an extensive villous papilloma through the sigmoidoscope is neither easy nor without risk, and resection and anastomosis will probably give a better result. In this case the difficulty of adequate follow-up and the presence of diverticulitis made resection clearly indicated.

Two patients had a resection on account of multiple polyps being found at laparotomy, and the history of one is of interest.

Case 12.—Mr. H. H., aged 62 years, complained of passing black stools. Sigmoidoscopy showed blood in the upper rectum, and there was a filling defect in the sigmoid shown by a barium enema. At laparotomy several polyps could be felt and it was thought to be wise to resect the sigmoid loop and perform an anastomosis.

The opened specimen showed three pedunculated polyps 1–2 cm. in diameter, and four small sessile polyps. Section of two of the pedunculated polyps demonstrated malignant change, with early invasion of the stalks with adenocarcinoma of low-grade malignancy. The only lymph-node found was free of tumour. Three years later the patient was well.

3. Polyps which Present Difficulties in Treatment.—It has become clear that, for some polyps, there is more than one possible line of treatment. The exercise of choice in these cases will call for careful judgement. In some patients the considerations will be purely technical, depending on what can safely be accomplished in the individual, but in others the debate will centre on the pathological nature of the polyp and the extent of excision which will be necessary.

The problem of the extent of resection would be simplified it if were possible to distinguish on sight between benign and malignant polyps. This cannot be done and their nature is only known after removal and microscopy. The problem could be solved by a general use of radical operations, but there is a proper reluctance to do this when half the tumours will prove to be benign. The usual procedure is to carry out a local excision of what may be a malignant tumour and to examine the specimen. In most cases this shows that an adequate operation has been done, but when carcinomatous change is found there must be careful consideration of the treatment; occasionally a second and more radical operation is necessary. In this assessment of the doubtful case the work of Lockhart-Mummery and Dukes (1952) gives valuable guidance.

They point out that there must be detailed examination of both tumour and stalk. If malignant change is present this may be restricted to the mucosa, when it is termed 'focal carcinoma'. If there is invasion of the stroma, 'invasive carcinoma' is present (*Figs.* 162, 163). They make a point of assessing the degree of differentiation of the tumour, and on this basis they classify carcinomata as being of low, average, or high-grade malignancy.

The stalk of the tumour is examined and declared either free of tumour or invaded. If invasion is present, special note is taken of the proximity of the invasion to the line of section. If there is only a

narrow margin free of growth then the risk of mucosal recurrence is correspondingly increased. On the whole, this risk can be fairly easily assessed.

It is much more difficult to assess the risk of recurrence due to lymphatic metastasis, for here tumour emboli will have passed down the lymphatics in the stalk and there will not necessarily be histological evidence of this. It may appear that complete excision has been obtained when in fact a metastasis is lying in an adjacent lymph-node.

FIG. 162.—Focal carcinoma.

FIG. 163.—Invasive carcinoma.

Areas of carcinomatous change are shown in *Figs.* 162, 163 as solid black shading.
(*Redrawn from Lockhart-Mummery and Dukes* (1952).)

The report of Lockhart-Mummery and Dukes shows that a polyp containing 'focal carcinoma' can be safely treated by local removal. This treatment cannot usually be considered adequate if 'invasive carcinoma' is present, although a polyp in which invasion is limited and the growth of low-grade malignancy will probably be well treated by local excision only. It is, however, clear that, once significant invasion by carcinoma of average-grade malignancy has occurred, a radical resection must be done if early recurrence is to be avoided. This policy will lead to a certain number of unnecessary operations (*Case* 10), but it will also result in a metastasis being removed when there is no significant infiltration of the bowel wall, as in *Case* 9. The experience of Scarborough and Klein (1948) is relevant here. In a series of 37 colonic polyps there were 6 examples of 'invasive carcinoma'. All 6 patients had a radical resection and in 3 there were metastases in mesenteric lymph-nodes; in none of these patients was there any invasion of the stalk of the polyp by growth.

Enough has been said to show that the correct line of treatment will only be chosen after the weighing of all available evidence.

SUMMARY AND CONCLUSIONS

Although a diagnosis is easily reached in most patients with a polyp of colon or rectum, there are some in whom considerable persistence in investigation is needed. The importance of altered blood seen on the mucosa at sigmoidoscopy is stressed.

Treatment will vary with the site of the primary tumour.

Polyps in the colon, beyond the range of the sigmoidoscope, must be removed at laparotomy. The merits of local polypectomy and resection of bowel have been discussed. A general preference for resection is expressed, especially when there is suspicion of carcinomatous change or the poylps are sessile or multiple.

Polyps in the lower sigmoid and upper rectum require careful scrutiny and biopsy. Those with a long stalk are generally removed with the diathermy snare, and for the others resection must be considered.

Polyps in the lower rectum will be treated by local removal wherever possible, as the alternative may be excision of the rectum.

The treatment of difficult cases with malignant change is discussed.

Acknowledgements.—These patients, with the exception of *Case* 7, which is not included in the series, were all under the care of Mr. O. V. Lloyd-Davies. I wish particularly to thank him for permission to quote from the records, to undertake the treatment of some of these patients, and for his advice during the preparation of the article. Mr. Hugh Dudley and Mr. W. W. Richardson gave valuable criticisms of the original draft. I wish to thank the radiologists of the Middlesex Hospital, especially Dr. C. G. Whiteside, for their ready help. Mr. M. Turney, Middlesex Hospital Photographic Department, and Mr. W. Topp, Department of Medical Photography, University of Aberdeen, kindly prepared the photographic illustrations.

REFERENCES

DEDDISH, M. R., and HERTZ, R. E. (1957), *Surg. Clin. N. Amer.*, **37**, 1287.

LLOYD-DAVIES, O. V., MORGAN, C. NAUNTON, and GOLIGHER, J. C. (1953), in *British Surgical Practice* 1953: *Surgical Progress* (ed. Sir Ernest Rock Carling and Sir James Paterson Ross). London: Butterworth.

LOCKHART-MUMMERY, H. E., and DUKES, C. E. (1952), *Lancet*, **2**, 751.

SCARBOROUGH, R. A., and KLEIN, R. R. (1948), *Amer. J. Surg.*, **76**, 723.

SPONTANEOUS PERFORATION OF THE NORMAL PELVIC COLON

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SPONTANEOUS perforation of the pelvic colon in the absence of any pathological findings has been but rarely reported, and in a search of the literature I have been able to find only 13 cases.

Two cases are reported here, the first of which appears to be unique in that spontaneous perforation of the pelvic colon occurred on two occasions, with an interval of 3 years between the episodes.

CASE REPORTS

Case 1.—

FIRST ADMISSION.—The patient, a male aged 54 years, was admitted to the Royal Free Hospital under the care of Mr. Rodney Maingot on Sept. 4, 1954, having had a sudden severe lower abdominal pain during the act of defæcation six hours before. He had previously suffered from chronic constipation, and had not had his bowels open for five days before admission. The pain had increased in intensity, but there had been no nausea or vomiting. His previous health had been good.

ON EXAMINATION.—A well-covered man, who was obviously in severe pain, pale, and shocked. T. 97·6° F., P. 110, B.P. 100/60. The tongue was dry and coated.

There was generalized rigidity of the abdomen, with extreme tenderness in the lower abdomen. Bowel-sounds were absent. There was no evidence of hernia. Rectal examination showed diffuse tenderness. A diagnosis of a perforated viscus was made, and laparotomy was performed.

AT LAPAROTOMY.—The abdomen was opened through a left paramedian incision, and there was found to be a mass of formed fæces in the pelvis. Examination of the pelvic colon disclosed a linear perforation, 1½ in. long, on the anti-mesenteric border. The perforation had the appearances of a tear, the edges being clean and the mucosa seeming healthy. The remainder of the colon was quite normal, there being no evidence of diverticulosis or other disease. No abnormality was found elsewhere in the abdomen.

The perforation was sutured in two layers, and a transverse colostomy was performed.

PROGRESS.—Post-operatively the patient was treated with continuous gastric suction and intravenous therapy. Intravenous terramycin was given. On the thirteenth post-operative day a swinging pyrexia developed, and X-ray examination showed elevation of the right diaphragm, which, with a white-cell count of 18,7000 (83 per cent polymorphs), suggested a subphrenic infection. This was treated conservatively with antibiotics and the infection subsided.

Ten weeks after the original operation the colostomy was closed. Sigmoidoscopy at this time showed no evidence of disease of the lower bowel, and a subsequent barium enema was also normal.

SECOND ADMISSION.—The patient, now aged 57 years, was re-admitted on Dec. 21, 1957, with a history that whilst getting out of bed five hours previously he had felt a sudden pain in the left iliac fossa which rapidly became more severe and spread to the rest of the abdomen. He had felt sick but had not vomited.

He had been quite well since his discharge from hospital three years previously, apart from chronic constipation for which he regularly took liquid paraffin. He had, however, taken no stronger aperient.

ON EXAMINATION.—The patient was in severe pain and was pale but not sweating. T. 97° F., P. 80, B.P. 140/90.

There was guarding and tenderness over the whole abdomen, with rigidity in both lower quadrants. Bowel-sounds were absent. There was no hernia.

A diagnosis of perforated colon was made, and laparotomy was performed.

AT LAPAROTOMY.—A lower left paramedian incision was made, and when the peritoneum was opened free fluid fæcal matter was found. A hard scybalous mass of fæces was found lying in the rectovesical pouch.

In the pelvic colon there was a longitudinal perforation 1 in. in length on the antimesenteric border. The margins of the perforation were quite healthy and the mucosa appeared normal and was pouting through the perforation. The colon was not distended but contained numerous scybalous masses. No disease was found in the colon or elsewhere in the abdomen.

The loop of pelvic colon with the perforation was exteriorized as a loop colostomy through an incision in the left iliac fossa, and after toilet of the peritoneum had been carried out the laparotomy wound was closed.

During the operation one pint of blood was given, and this was followed by a dextrose-saline infusion which was continued during the post-operative period. Intravenous terramycin was given.

Progress was slow but satisfactory until the sixth post-operative day, when the patient developed an acute left-sided parotitis which responded poorly to antibiotics and which resulted in œdema of the larynx. Emergency tracheostomy was performed under local anæsthesia, but the patient died of bronchopneumonia on the ninth day.

POST-MORTEM EXAMINATION.—The immediate cause of death was found to be bronchopneumonia, with a left empyema. There was also a small left subphrenic abscess.

The Colon.—Several small superficial stercoral ulcers were present, involving only the mucosa.

Above the perforation in the pelvic colon was a small area of scarring on the serosal surface, which was thought to represent the site of the previous perforation.

Just below the perforation the wall of the pelvic colon showed a rather ill-defined out-pocketing in which the wall of the bowel was much thinner than the rest of the colon. The perforation had, however, occurred in that part of the bowel which was of normal thickness.

No other abnormality was found in the bowel.

Case 2.—A male patient, aged 76 years, was admitted to the Royal Free Hospital under the care of Mr. Emlyn Williams on Jan. 22, 1959, complaining of lower abdominal pain of sudden onset twelve hours previously. The pain began soon after getting out of bed in the morning and was extremely severe. He had vomited several times.

There was a previous long history of chronic constipation, and prior to admission his bowels had not been opened for two days.

ON EXAMINATION.—The patient was pale and restless. T. 102° F., P. 108, B.P. 140/75. The abdomen was rigid in both iliac fossæ, with marked rebound tenderness. There was no evidence of hernia. A diagnosis of a perforated viscus was made and laparotomy performed.

AT LAPAROTOMY.—The abdomen was opened through a lower right paramedian incision, and a small quantity of free purulent fluid was found. A scybalous mass of fæces 1½ in. by 1 in. was found lying in the rectovesical pouch. Examination of the colon revealed a perforation 1 in. in length just above the pelvirectal junction. This was on the antimesenteric border and had the appearances of a tear, the edges of the perforation showing no signs of inflammation. There was no associated diverticulum.

The descending colon contained scybala, but was not distended.

The perforation was sutured and a transverse colostomy was performed.

Post-operatively his condition steadily deteriorated in spite of intravenous therapy, continuous gastric suction, and intravenous terramycin, and he died 48 hours later.

AETIOLOGY

Perforation of a simple ulcer or necrotic lesion of unknown aetiology is a well-recognized clinical entity, and must be differentiated from the type of perforation described here. Following perforation of a simple ulcer there are obvious acute inflammatory changes in the bowel wall adjacent to the perforation, whereas in the true spontaneous perforation the bowel wall appears normal.

Table I.—Summary of Cases

Author		Age	Sex	Situation	Constipation	Scybalous Mass	Time of Perforation	Notes
Brown (1944)		30	M.	Pelvirectal	No	"Small quantity of fæces in pelvis on rising"		
Hoffert (1946)		63	M.	Pelvic colon (anti-mesenteric)	Yes	In pelvis	?	
Weinstein and Roberts (1952)		63	M.	Pelvic colon (anti-mesenteric)	Yes	In pelvis	At stool	
Serjeant and Raymond (1952)		67	F.	Pelvirectal	No	In pelvis	7 a.m.	Followed barium meal
Brearley (1954)		68	M.	Pelvic colon (anti-mesenteric)	Yes	In pelvis	?	Followed alkaline medication
Eadie (1955)		57	F.	Pelvic colon (anti-mesenteric)	Yes	In pelvis	At stool	
Birbis (1955)	1.	38	M.	Pelvirectal	Yes	In pelvis	At stool	
	2.	69	M.	Pelvirectal	Yes	In pelvis	At stool	
	3.	63	M.	Pelvic colon (anti-mesenteric)	Yes	In pelvis	At stool	
Henderson (1957)	1.	38	F.	Pelvic colon (anti-mesenteric)	?	Plugging perforation	?	
	2.	49	F.	Pelvirectal	Yes	In pelvis	Early morning	
Rapp (1958)	1.	69	F.	Pelvic colon (right side)	Yes	In pelvis	During night	
	2.	65	F.	Pelvic colon (anti-mesenteric)	Yes	In pelvis	?	

Post-mortem Examination.—The immediate cause of death was bronchopneumonia and peritonitis.

The Colon.—The perforation was situated 3 in. above the pelvirectal junction and was across the anterior tænia coli. It was not associated with a diverticulum. The colonic and rectal mucosæ were somewhat œdematous but not obviously inflamed.

PREVIOUSLY REPORTED CASES

Wilensky and Kaufman (1937) collected 33 cases of spontaneous rupture of the bowel in the presence of hernia, and 1 of these (Hahn, 1931) was in the large bowel. It has been thought advisable to exclude this case from the present series, and also that of Lataix (1949) in which a ventral hernia was present, since the relationship between hernia and perforation of the bowel is well recognized, having been first described by Bunge (1905).

Two cases have, however, been included in which a contributing factor was recognized, since it would appear that the mechanism of the rupture was the same as in the other cases in this series. These cases are described by Serjeant and Raymond (1952) and Brearley (1954). The former was attributed to barium in the colon after a barium meal and the latter to the presence of masses of alkali in the colon.

Of the cases collected some occurred during the act of defæcation and some otherwise, but there seems no reason not to regard the former as spontaneous perforations.

The cases collected from the literature are summarized in *Table* I.

Cases of perforated simple ulcer have been described by Roux and Lenoel (1944), Sicard, Picard, and Lachartre (1956) and others, and a similar case has been seen recently in the Royal Free Hospital in a man of 56 years who was admitted under the care of Mr. G. Qvist with a history of acute generalized abdominal pain of 24 hours' duration. At laparotomy there was a 1-in. perforation in the middle of the pelvic colon, and the adjacent bowel appeared gangrenous. The pelvic colon was excised and on examination showed acute inflammatory changes in the bowel wall surrounding the gangrenous patch.

In 5 of the cases collected here the site of rupture is at the pelvirectal junction. Wilensky and Kaufman (1937) state that rupture at this site occurs predominantly in women, when it is associated with rectal prolapse. Rupture usually occurs at stool, and it is associated with prolapse of the small bowel into the lumen of the rectum. In the 5 cases in this series this does not seem to have been the mechanism, since fæcal matter was extruded from the colon, and no small bowel entered the rectum. Furthermore, there was no rectal prolapse, and 3 of the cases were male patients. It would seem probable therefore that the aetiology in these 5 cases is the same as in those in which the pelvic colon itself is affected.

It has been suggested that the bowel wall ruptures at a site where the muscle coats are abnormally thin,

as is sometimes found in the prediverticular stage of diverticulitis (Edwards, 1939). However, as pointed out by Eadie (1955), rupture of diverticula rarely occurs in the absence of inflammation. It is also of interest to note that in the first case reported here an area of abnormal thinness of the muscle coats was in fact present, but the rupture of the bowel occurred not at this point but above it, where the wall was normal.

Brearley (1954) suggested that impaction of a hard fæcal mass on the antimesenteric wall of the bowel, where the blood-supply is poorest, may lead to an ischæmic necrosis and subsequent perforation.

Birbis (1955) has carried this theory a stage further. He observed that in the 3 cases reported by him a large fæcolith was present either in the pelvis or plugging the gap in the bowel wall. He postulates a 'hernia of weakness' in the wall of the bowel, produced partly by repeated straining at stool and partly by accumulation of a fæcolith, this leading eventually to a perforation.

It would appear that this theory is in accordance with all the facts observed in the collected cases.

In particular, a scybalous mass of fæces is specifically mentioned to have been lying free in the pelvis or plugging the perforation in all the cases except that of Brown (1944), in which there was "a small quantity of fæces" in the pelvis. There is also a history of chronic constipation in all the cases except three, a condition predisposing to the formation of a hard fæcal mass.

In all cases in which the time of perforation has been recorded it has occurred either at stool, when there would be increased strain on the bowel wall, or early in the morning, when the colon may be expected to be loaded with fæces.

All the perforations except one occurred on the antimesenteric border of the colon, where the blood-supply is poorest, and not in the position where colonic diverticula commonly occur.

SUMMARY

Two cases of spontaneous perforation of normal pelvic colon are reported. In one case there were two perforations with an interval of three years between the episodes.

Thirteen similar cases have been collected from the literature, and their salient features tabulated.

Various theories concerning the aetiology of this condition are discussed, and it is concluded that that of Birbis (1955) is in accordance with the facts observed in all these cases.

Addendum.—Since this paper was prepared a further case has been reported (Cronin, 1959).

A female patient, aged 69 years, sustained a spontaneous rupture of the pelvic colon in the absence of hernia or disease of the bowel. At operation there was found to be a linear tear 4 cm. long on the antimesenteric border at the apex of the pelvic colon. Lumps of fæces were lying free in the pelvis, and the descending colon was filled with hard fæces.

I should like to thank Mr. Rodney Maingot, Mr. George Qvist, and Mr. Emlyn Williams for permission to report these cases; also Mr. Maingot for his help and advice in the preparation of this paper.

REFERENCES

BIRBIS, M. P. (1955), *Bordeaux Chir.*, **1**, 17.
BREARLEY, R. (1954), *Brit. med. J.*, **1**, 743.
BROWN, J. (1944), *Brit. J. Surg.*, **31**, 307.
BUNGE (1905), *Beitr. klin. Chir.*, **47**, 771.
CRONIN, K. (1959), *Brit. J. Surg.*, **47**, 43.
EADIE, J. W. (1955), *Ibid.*, **43**, 189.
EDWARDS, H. C. (1939), *Diverticula and Diverticulitis of the Intestine*. Bristol: John Wright.
HAHN, H. (1931), *Zbl. Chir.*, **58**, 469.
HENDERSON, R. G. (1957), *Brit. J. Surg.*, **44**, 383.
HOFFERT, H. E. (1946), *Minnesota Med.*, **29**, 935.
LATAIX, P. (1949), *Sem. Hôp. Paris*, **25**, 2279.
RAPP, C. R. (1958), *Canad. J. Surg.*, **1**, 157.
ROUX, M., and LENOEL, J. (1944), *J. Chir., Paris*, **60**, 20.
SERJEANT, J. C. B., and RAYMOND, J. A. (1952), *Lancet*, **2**, 1245.
SICARD, A., PICARD, J., and LACHARTRE, P. (1956), *Mém. Acad. Chir., Paris*, **82**, 765.
WEINSTEIN, M., and ROBERTS, M. (1952), *J. Amer. med. Ass.*, **149**, 1016.
WILENSKY, A. O., and KAUFMAN, P. A. (1937), *Ann. Surg.*, **106**, 373.

PEPTIC ULCER AS ENCOUNTERED IN A SURGICAL UNIT IN SOUTH INDIA

A REVIEW OF 198 CASES

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POPULATION surveys are essential in order to appreciate the special characteristics of indigenous diseases in an underdeveloped country. These studies require adequate personnel and considerable time and money. Quick results cannot be expected. Such a survey has recently been started at the Christian Medical College, Vellore, and it is hoped that eventually valuable information about the true incidence of duodenal ulcer and its complications in that area will be obtained. In the meantime, hospital statistics provide the only figures that are available.

Mass hospital statistics based on both in-patients and out-patients have been of value in defining the sex and age incidence and the gastric-duodenal ulcer ratio in areas of the world with an adequate supply of hospitals and general practitioners. These conditions do not apply in India and the significance of hospital statistics suffers accordingly. There has always been great difficulty in acquiring adequate information from out-patients owing to the overcrowding of these clinics and inadequate follow-up of the cases. It is

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probably for this reason that most workers have excluded out-patient figures (Somervell and Orr, 1936; Dogra, 1940; Menon, 1940; Lal, 1950; Hadley, 1957) and this further reduces the significance of such work.

Work based on personal series of cases also has severe limitations, but such findings are more reliable sources of detailed information than those based on mass hospital statistics. I have therefore decided to present my findings in this small series of 198 personal cases with similar exclusion of out-patient statistics.

Table I.—Classification of Cases

	Present Series: 198 Cases	Medical College, Vellore: 350 Surgical Cases 1955–6 (Hadley, 1957)	General Hospital, Madras: 826 Surgical Cases (Menon, 1940)
Duodenal ulcer	153 (77·3 per cent)	280 (80 per cent)	670 (81·1 per cent)
Combined duodenal and gastric ulcer	18 (9·1 per cent)	13 (3·7 per cent)	79 (9·6 per cent)
Gastric ulcer	12 (6·1 per cent)	22 (6·3 per cent)	77 (9·3 per cent)
Stomal ulcer	15 (7·5 per cent)	14 (4·0 per cent)	Not included
Miscellaneous	—	21 (6·0 per cent)	—

I feel that this paper may be of value in emphasizing certain as yet undescribed features of duodenal ulcer encountered in South India.

METHODS

I have reviewed all the case sheets of patients who have been under my care in one of the three general surgical firms in this hospital between August, 1954, and February, 1958, inclusive. I have excluded ulcer cases with free perforation and those patients who came into my surgical wards but were discharged without operation (both very small groups). This series comprises all cases that I operated on from August, 1954, to June, 1957, but the cases from July, 1957, to February, 1958, are consecutive cases treated in my firm (Firm II). I operated on the majority of this latter group of cases, but all operation findings during this period were classified according to a mutually agreed plan so that the information could be entered on edge marking cards.

CLASSIFICATION

These cases are classified in *Table I* and comparisons made with larger groups of cases reported from this hospital (Hadley, 1957) and from the General Hospital in Madras (Menon, 1940). A special interest in the identification and recording of all ulcers and scars may have accounted for the higher incidence of combined gastric and duodenal ulcers in my series compared with the larger series reported from all three general surgical firms of this hospital by Hadley (1957). The higher incidence of stomal ulcers in my series was directly related to the start, in 1957, of a follow-up survey of all cases of peptic ulcer treated in this hospital over the past ten years. All these cases had originally been treated by gastro-enterostomy more than three years previously. Up till now no stomal ulcer has been found at operation following gastro-enterostomy with vagotomy, partial gastrectomy, or segmental resection.

A. DUODENAL ULCERS

There were 153 cases of duodenal ulcer in this series.

Table II.—Age and Sex

	Present Series: 153 Duodenal Ulcers	Present Series: 198 Peptic Ulcers	Christian Medical College, Vellore: 406 Medical and Surgical Peptic Ulcers (Hadley, 1957)	General Hospital, Madras: 1410 Duodenal Ulcers (Menon, 1940)
Sex: Males	142 (92·0 per cent)	184 (93·0 per cent)	379 (90·3 per cent)	1359 (96·4 per cent)
Females	11 (8·0 per cent)	14 (7·0 per cent)	27 (6·7 per cent)	51 (3·6 per cent)
Age at diagnosis: 25–44	89 (58·1 per cent)	98 (45·4 per cent)	242 (59·8 per cent)	Age 20–39: 978 (69·4 per cent)
45–54	31 (20·3 per cent)	41 (20·7 per cent)	84 (20·7 per cent)	Age 40–49: 237 (16·8 per cent)
				Age 50–59: 63 (4·5 per cent)

Sex and Age Ratio.—These are shown in *Table II*, where comparisons are also made as in *Table I*. The large number of males is characteristic of all series reported from South India, and this is probably significant as, apart from Moslems, women readily attend hospital for medical and surgical care. The small number of female surgical beds does not account for the marked male preponderance. Thus during the years 1932–6, when only women were treated in the C.M.C. Hospital, Vellore, there were 16,470 admissions. Of these cases there were only 56 with peptic

ulcer, i.e., 0·03 per cent of the total admissions. In 1956 the peptic ulcer admission rate to this hospital was 2·5 per cent and this has been fairly constant over the whole period since male wards have been established.* This rise in incidence correlates well with the expected rise calculated from the sex incidence.

The majority of my cases were under 44 years of age and these included 3 cases under 12 years, including a boy aged 4 years with a chronic duodenal ulcer confirmed at operation. The age and sex ratios correspond closely with those in Madras.

Pain and Vomiting (*Table III*).—Severe pain was a frequent complaint. In 37 per cent of the duodenal ulcer patients in my series severe back pain

Table III.—Pain and Vomiting

	153 Duodenal Ulcers Present Series	826 Duodenal Ulcers, Madras (Menon, 1940)
Aggravation of pain by food	86 (56·2 per cent)	Frequent
No mention in notes	57 (37·2 per cent)	
Relief of pain by vomiting	111 (72·5 per cent)	Very frequent
No mention in notes	5 (3·2 per cent)	
Vomiting	136 (88·8 per cent)	Over 75 per cent
Induced	65 (42·4 per cent)	Frequent

was a prominent feature, but in an equal number of cases this type of pain had not been definitely excluded in the case notes. Aggravation of pain by food and relief by vomiting were frequent features. Vomiting was a very common complaint. It was considered markedly stenotic in type (i.e., frequent vomiting of large amounts of stale food) in 40 out of the last 65 cases.

Bleeding.—This was a most difficult factor to assess from the history, and this is probably due to the confusion in the description of colour that is met with in the common languages in this area. Hæmatemesis and melæna are generally thought to be rare in South India (Somervell and Orr, 1936; Hadley, 1957). Of the 153 duodenal ulcer cases, 13 gave a clear history of previous hæmatemeses and in another 13 there was a fairly definite history of previous melæna. Streaking of vomitus with blood was a fairly frequent complaint and was probably due to minor pharyngeal trauma during induction of vomiting. The dirty vomitus of gross retention may be confused with true hæmatemesis when the history is taken. Hæmorrhage is only rarely the cause for admission. It is hoped that the population survey will lead to better understanding of the true frequency of this complication.

* Several large specialty units (Thoracic Surgery, Neurosurgery, Gynæcology, and Pædiatrics) mask the high frequency of peptic ulcer cases in the general surgical wards of this hospital.

Diagnosis of Gastric Retention (*Table IV*).—The diagnosis of stenosis and gastric retention was mainly based on a definite clinical history of repeated vomiting of stale food. The large number of these cases which also had prominent visible peristalsis is shown in the table. The relatively small percentage

Table IV.—Gastric Retention

	153 Duodenal Ulcers Present Series	245 Duodenal Ulcers, Madras (Dogra, 1940)
Visible peristalsis	74 (48·3 per cent)	165 (67·3 per cent)
No mention in notes	18 (11·7 per cent)	—
Delay on 85 barium meals	39 (45·8 per cent)	—
Diagnosis of stenosing duodenal ulcers	130 (84·9 per cent)	—
History suggestive of marked obstruction from onset	(23 out of last 65 cases—35·4 per cent)	—

of cases with delay on barium meal examination is due to the fact that this investigation was usually dispensed with once visible peristalsis had been unequivocally demonstrated. The large proportion of cases with evidence of stenosis is characteristic of all series of duodenal ulcer cases from South India (Bradfield, 1927; Somervell, 1927; Somervell and Orr, 1936; Kini and Ras, 1939; Menon, 1940; Dogra, 1940; Lal, 1950; Hadley, 1957). A similar high incidence of stenosis is reported in Southern Nigeria (Ellis, 1948; Konstam, 1954).

Operative Findings.—During the period from July, 1957, to February, 1958, a determined effort was made in my surgical firm to collect detailed operative measurements from consecutive cases of duodenal ulcer. There is a widespread impression that measurements made at the time of operation are unreliable. This is probably based on the assumption that "direct inspection may lead to error in the localisation of duodenal ulcer" (Moynihan, 1910). Hurst and Stewart (1929) stated, "Radiological evidence is of much greater value in this connection than observations made during operations". However, I have found that careful mobilization (Kocherization) of the second part of the duodenum allows

Table V.—Findings in 50 Consecutive Cases of Peptic Ulcer

	No. of Cases
'Distal' duodenal ulcer or scar*	33
Dilated pyloric ring	33
Combination of dilated pyloric ring and 'distal' duodenal ulcer or scar	32

* Ulcers situated 2 cm. or more from pyloric ring.

measurements to be made between the palpable pyloric ring and the ulcer. These were made with a short sterile transparent ruler laid alongside the first part of the mobilized duodenum. These extra procedures have caused no additional post-operative complications and there has been no operative mortality during this period. Sixty-six per cent of the cases had a 'distal' duodenal ulcer or scar (*Table V*).

The word 'distal' refers to lesions situated 2·0 cm. or more from the pyloric ring.

A wide pyloroplasty was carried out as part of the operative procedure in 26 of these 50 consecutive cases (Group A). One case was treated by pyloroplasty combined with vagotomy while segmental resection combined with pyloroplasty (Wangensteen, 1952), was carried out on the remaining 25 cases*. The opportunity was taken to inspect the ulcer craters during the performance of the pyloroplasty and to measure the size of the ulcer and its distance from the pyloric ring. This latter structure was found to be a much better landmark than the so-called 'pyloric vein'. The findings in this group of cases are shown in *Tables VI* and *VII*. In the remaining 24 cases the

Table VI.—SITE OF 'DISTAL' ULCERS IN GROUP A

Distance from the Pyloric Ring of the Most Distally Situated Duodenal Ulcer or Scar	*Cases with Single Ulcer*	*Cases with Multiple Ulcers*
2·0 cm.	5	1
2·5 cm.		3
3·0 cm.	2	2
3·5 cm.		1
4·0 cm.	1	1
5·0 cm.		3

Total cases with 'distal' ulcer(s) = 19, out of 26 cases in Group A.

Table VII.—SIZE OF ULCERS IN GROUP A CASES—30 ACTIVE ULCERS IN 26 CASES

Size of Ulcers	*No. of Cases*
0·4 cm. and under	17
0·5 cm.–0·9 cm.	4
1·0 cm.–1·9 cm.	6
2·0 cm. and over	3
Total	30

site of the duodenal ulcer or scar was carefully determined by inspection and palpation. The high percentage of 'distal' lesions is emphasized and these were well correlated with clinical evidence of gastric retention. The importance of opening the duodenum is shown in *Table VIII*. This is the only way to obtain accurate data about a duodenal ulcer. It has frequently been said that, in South India, the ulcer is

Table VIII.—SITE OF DUODENAL ULCERS (50 CONSECUTIVE CASES)

	DUODENUM OPENED (26 cases) 35 DUODENAL ULCERS OR SCARS SEEN	DUODENUM UNOPENED (24 cases) 24 DUODENAL ULCERS OR SCARS FELT
Posterosuperior	19	5
Anterosuperior	6	3
Posterior	2	4
Anterior	8	12

on the anterior wall of the duodenum (Somervell and Orr, 1936; Dogra, 1940; McLaren, 1955), and this was the usual operation report in my cases when the duodenum was unopened. This was not the case in the group in which the duodenum was opened, when the majority of the ulcers were found to be situated posterosuperiorly.

* In the present series of cases the antrum was anastomosed to a small residual fundic pouch after excision of three-quarters of the acid-secreting area of the stomach.

It is interesting that Ellis (1948) was impressed with the frequency of 'distal' duodenal ulcer combined with a dilated pyloric ring in his surgical cases in Nigeria. No measurements, however, were made, and more recent reports from this area (Joly, 1956; Konstam, 1957) do not refer to these findings.

Correlation with Radiographic Findings.—A retrospective analysis of the radiographs that were carried out in this series of cases has been made and the results reported (Paterson and Hancock, 1958). There was clear evidence of post-bulbar ulceration in 20 per cent of the present series of cases and the clinical features in all of them were those of active obstructing duodenal ulcers. Visible peristalsis was unequivocally demonstrated in 6 of these 10 patients. A careful analysis of 1803 consecutive barium meal examinations at Vellore (including 527 cases of duodenal ulcer) has now been carried out (Paterson and Hancock, 1959). Of these cases 24 per cent had post-bulbar ulcers. Now, these lesions are relatively rare in Western countries, probably well below 5 per cent of all radiologically defined duodenal ulcers or scars. However, it is difficult to make comparisons owing to the large number of variable factors.

B. GASTRIC AND COMBINED GASTRODUODENAL ULCERS

The cases of gastric ulcer and combined gastric and duodenal ulcer in this series are too few in number to be considered in detail. In general, it was found that the combined gastric and duodenal ulcer cases closely resembled the duodenal ulcer cases clinically except for two aspects: (1) Bleeding was twice as common in this group as in the pure duodenal ulcer cases. (2) A large number of these cases gave a history of obstructive symptoms from the onset of their complaints.

It is interesting that 5 of the 12 gastric ulcers and 2 of the 18 combined gastric and duodenal ulcers had craters in the stomach which were 4·0 cm. or more in greatest diameter, including one ulcer of 15·0 cm. and one of 10·0 cm. maximum diameter. These 7 cases could thus be classified as having 'giant ulcers' (Turner, Dockerty, Priestley, and Comfort, 1957).

C. STOMAL ULCERS

The number of stomal ulcers diagnosed varied directly with the interest of the surgeon and radiologist concerned, and the frequency of this condition markedly increased after the commencement in 1957 of a follow-up survey of all peptic ulcer cases treated in this hospital during the past ten years. A study is now being made of over 70 stomal ulcers seen in this hospital (Keswani and Paterson, 1958). The frequent argument that "anastomotic ulcers are uncommon in this area and therefore simple gastro-enterostomy is the treatment of choice" has no scientific foundation. If these patients are carefully looked for they will be found in any large series treated by simple gastro-enterostomy without vagotomy, provided such patients are followed up long enough.

Operations and Operative Mortality.—These are shown in *Table IX*. It will be noted that there was no mortality in the group treated by segmental resection. The patient treated by pyloroplasty with suture had a massive hæmatemesis from a giant duodenal ulcer measuring 4·5 cm. in maximum

diameter. The mortality in the 99 cases treated by partial gastrectomy is high. All the deaths occurred during my first year at Vellore, and I have had no personal mortality since August, 1955, in over 70 partial gastrectomies for peptic ulcer. Three of the 5 patients died because of an error of judgement. In 2 of them gastroduodenectomy had been carried out for distally situated duodenal ulcers. I have since employed closure of the duodenum proximal to the obstructing ulcer in these cases without complications. In the third, a giant simple gastric ulcer was mistaken for a locally advanced carcinoma and treated by near-total gastrectomy, right hemicolectomy, splenectomy, and excision of the body and tail of the pancreas. A localized subphrenic abscess was found at post-mortem. The cause of death in the remaining 2 cases was (1) pulmonary embolism following gastrectomy for a juxta-œsophageal ulcer, and (2) cardiac arrest which occurred at the time of removal of the endo-tracheal tube.

Table IX.—Operations and Operative Mortality

Operations		Mortality
Partial gastrectomy	99	5
Segmental resection with pyloroplasty	53	0
Vagotomy and gastro-enterostomy	36	0
Gastro-enterostomy (including 3 children under 12)	9	0
Pyloroplasty with suture of bleeding ulcer	1	1
Total	198	6

DISCUSSION

Duodenal ulcer patients in South India present in hospital with a similar pattern of symptoms. There also appear to be similarities between India and Southern Nigeria (Konstam, 1957). Dogra (1940) stated that the number of operations on the stomach suddenly increased by 39 and 56 times respectively, in the General Hospital, Madras, and the London Mission Hospital, Neyoor, "following the arrival and pioneer work of two great surgeons in South India, namely Bradfield and Somervell in Neyoor". The interesting thing is, that following this dramatic start, the incidence of stenosis and gastric retention remained uniformly high in Neyoor up to 1942 (Somervell, 1942). Similarly in Vellore a survey has shown little change in the stenotic rate amongst duodenal ulcer cases over the last five years. This is an unexpected finding because there has been a definite increase in ulcer consciousness and acceptability of surgery in the patients whom we treat. It would be expected that there would be a comparable decrease in the incidence of stenosis, but this has not yet been reported. The paucity of general practitioners in the villages, transport difficulties (Avery Jones, 1957), and late diagnosis of the disease in South India may account for this continued high stenotic rate in hospital surveys, despite the availability of good surgical treatment in a few hospital centres over the past thirty years. On the other hand, this constant incidence of stenosis may be a reflection of a true difference in presentation and complications of duodenal ulcer as it occurs in this part of the world.

Operative findings on a small consecutive series of cases during these eight months showed that a large number of cases (66 per cent) had 'distal' duodenal ulcers, i.e., ulcers situated 2·0 cm. or more from the pyloric ring. Now the general opinion is that the vast majority of duodenal ulcers in Western countries and in Japan occur within 2 cm. of the pyloric ring. This opinion is based on: (1) Autopsy findings (Hurst and Stewart, 1929; Portis and Jaffe, 1938; Magnus, 1954; Tokoro, 1957); (2) Radiological findings (Ball, Allen, and Golden, 1948; Carstairs, 1955; Ramsdell, Bartholomew, Cain, and Davis, 1957; Kurokawa and Masuda, 1957; and Cooke and Hutton, 1958). The percentage of 'distal' ulcers and post-bulbar ulcers was so much higher than might have been expected, and these lesions occurred so regularly, that the publication of these preliminary findings is justified. I have not been able to extend these observations over a larger series of cases as I had to leave India in March, 1958, but I hope that this paper will stimulate others to carry out a more thorough study of this distressing disease.

Comparable reports on operative measurements in consecutive cases of duodenal ulcer in Western countries are unavailable. Autopsy and radiological findings have been considered sufficient evidence, and surgeons have found no reason to uncover this difficult area at the time of operation, simply in order to make measurements. However, the radiologist frequently fails to demonstrate post-bulbar ulcers and autopsy findings are notoriously misleading in giving them a true picture of this disease. Thus Hurst and Stewart (1929) found that half their cases had healed lesions on post-mortem examination and they emphasized the fact that clinically pyloric obstruction rarely gives rise to symptoms unless active ulceration is present. There may be, therefore, important differences between post-mortem and operative measurements in cases of obstructing duodenal ulcer, and a similar survey of operative findings in other countries would be of value in the study of the geographical pathology of this disease. Such procedures need not add to the operative risk. Furthermore, the use of the pyloric ring as a landmark by the surgeon would result in a clearer definition of such terms as "pyloric channel ulcers" (Texter, Baylin, Ruffin, and Legerton, 1953) and of "ulcers at or near the pylorus" (Foulk, Comfort, Butt, Dockerty, and Weber, 1957). The pyloric ring is recognized as a sharply defined entity once it has been split open, and if a pyloroplasty is carried out as part of the operative procedure it should be possible for the surgeon to relegate the majority of these ill-defined ulcers to one or other side of the ring by palpation and inspection of the opened-up gastro-duodenal area.

SUMMARY

A personal series of 198 peptic ulcer cases is reviewed. The clinical features are comparable with those of larger series reported from this hospital and Madras.

All reports from South India show high incidences of: (1) Male patients, (2) duodenal as opposed to gastric ulcers, and (3) gastric retention. The significance of these factors is discussed.

Careful operative measurements were made in a series of 50 consecutive duodenal ulcer patients; 33 of them had 'distal' ulcers (ulcers situated 2·0 cm. or more from the pyloric ring) and 10 had post-bulbar ulcers. This high incidence of post-bulbar ulceration has been confirmed in an analysis of 527 consecutive barium meals which were reported as showing duodenal ulcers or scars (Paterson and Hancock, 1960).

A relatively large number of 'giant' duodenal ulcers and 'giant' gastric ulcers were encountered.

Operative measurements from other centres would be of value in the study of the geographical pathology of peptic ulcer.

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REFERENCES

BALL, R. P., ALLEN, L., and GOLDEN, R. (1948), *Amer. J. Roentgenol.*, **59**, 90.
BRADFIELD, E. W. C. (1927), *Trans. Far-East. Ass. trop. Med.*, **1**, 221.
CARSTAIRS, L. S. (1955), *Postgrad. med. J.*, **31**, 162.
COOKE, L., and HUTTON, C. F. (1958), *Lancet*, **1**, 754.
DOGRA, J. R. (1940), *Indian J. med. Res.*, **28**, 145.
ELLIS, M. (1948), *Brit. J. Surg.*, **36**, 60.
FOULK, W. T., COMFORT, M. W., BUTT, H. R., DOCKERTY, M. B., and WEBER, H. M. (1957), *Gastroenterology*, **32**, 395.
HADLEY, G. (1957), *Trans. 6th Mtg, Int. Soc. Geogr. Path., Paris*, 472. Basel: Karger.
HURST, A. F., and STEWART, M. S. (1929), *Gastric and Duodenal Ulcer*. London: Oxford University Press.
JOLY, B. M. (1956), *West Afr. med. J.*, **5**, 55.
JONES, F. A. (1957), *Brit. med. J.*, **1**, 719.
KESWANI, K. G., and PATERSON, D. E. (1958), personal communication.
KINI, M. G., and RAS, V. R. (1939), *Antiseptic*, **36**, 841.
KONSTAM, P. G. (1954), *Lancet*, **2**, 1039.
— — (1957), *Trans. 6th Mtg, Int. Soc. Geogr. Path., Paris*, 229. Basel: Karger.
KUROKAWA, T., and MASUDA, H. (1957), *Ibid.*, 479.
LAL, SANGHAM (1950), *Indian J. Surg.*, **12**, 291.
MCLAREN, D. S. (1955), "Health and Disease in the Kond Hills, India", M.D. Thesis, Edinburgh.
MAGNUS, H. A. (1954), *Postgrad. med. J.*, **30**, 131.
MENON, C. P. V. (1940), *Indian J. Surg.*, **2**, 5.
MOYNIHAN, B. G. A. (1910), *Duodenal Ulcer*. Philadelphia and London: Saunders.
PATERSON, D. E., and HANCOCK, D. M. (1958), *Brit. J. Radiol.*, **31**, 660.
— — — — (1960), *Ibid.*, **33**, 43.
PORTIS, S. A., and JAFFE, R. H. (1938), *J. Amer. med. Ass.*, **110**, 6.
RAMSDELL, J. A., BARTHOLOMEW, L. G., CAIN, J. C., and DAVIS, G. D. (1957), *Ann. intern. Med.*, **47**, 700.
SOMERVELL, T. H. (1927), *Trans. Far-East. Ass. trop. Med.*, **1**, 221.
— — (1942), *Brit. J. Surg.*, **30**, 118.
— — and ORR, I. M. (1936), *Ibid.*, **26**, 227.
TEXTER, E. L., BAYLIN, G. F., RUFFIN, T. M., and LEGERTON, C. W. (1953), *Gastroenterology*, **24**, 319.
TOKORO, Y. (1957), *Trans. 6th Mtg, Int. Soc. Geogr. Path., Paris*, 504. Basel: Karger.
TURNER, J. C., DOCKERTY, M. B., PRIESTLEY, J. T., and COMFORT, M. W. (1957), *Surg. Gynec. Obstet.*, **104**, 746.
WANGENSTEEN, O. H. (1952), *J. Amer. med. Ass.*, **149**, 18.

NEONATAL INTESTINAL OBSTRUCTION FROM A CONGENITAL HOLE IN THE MESENTERY

REPORT OF A CASE

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STRANGULATION of a loop of bowel through a slit or hole in the mesentery was a recognized form of internal hernia as long ago as 1885, when Treves discussed it in a Hunterian Lecture at considerable length. The condition is not excessively rare; reports of single cases have appeared from time to time: Hansmann and Morton (1939) refer to 38 cases in the literature prior to 1939 in a review of 467 internal herniæ. Cases usually present in adult life, and I have only been able to trace one other in an infant (Long, 1927). Gross (1953) refers to 3 cases seen in children, but gives no details, and does not include this in the differential diagnosis of neonatal intestinal obstruction. The following case is therefore worth placing on record.

CASE REPORT

S. B. (Hosp. No. 051010/59), born June 16, 1959, female, was the first child of a healthy mother whose pregnancy and labour had been normal. Though small (birth weight 5 lb. 15 oz.) the child seemed to be normal for the first 48 hours, taking feeds and passing meconium.

At 4 p.m. on June 18 she was seen to vomit and to be momentarily cyanosed. At 6 p.m. the abdomen was distended and visible peristalsis was present, but there were no other abnormal signs. By 9 p.m. the distension had not increased and the child's general condition remained unchanged over the next six hours, though small feeds of water were vomited.

Rectal examination showed no abnormality. The stool was examined microscopically, and found to contain squamous cells and lanugo hairs. A plain radiograph of

the abdomen (*Fig.* 164) showed distended coils of small bowel, but no fluid levels.

The pre-operative diagnosis was intestinal obstruction, probably at the level of the lower ileum, without suggestion of strangulation. The cause of the obstruction was a matter of conjecture: meconium ileus was excluded by the normal-looking stool which had been passed. Intestinal atresia was ruled out by finding hair and squamous cells in the stool. Malrotation of the small bowel was a possibility, but there was nothing in the radiograph to suggest it. At all events, laparotomy was indicated, and was performed at 3 a.m. on June 19, eleven hours from the first vomit.

Fig. 164.—Pre-operative radiograph showing absence of fluid levels and distended small bowel.

AT LAPAROTOMY.—

General Anæsthesia (Dr. John S. Toner).—After a sterilized rubber endotracheal tube (size OO) had been passed, anæsthesia was induced by a mixture of oxygen (2 l./min.), nitrous oxide (2 l./min.), and cyclopropane (300 ml./min.). Thereafter the proportion of cyclopropane was adjusted to give satisfactory operating conditions. Respiration was spontaneous, with occasional assistance. To assist closure 2·5 mg. of succinylcholine was given intramuscularly.

Operation.—A right paramedian incision was made. As soon as the peritoneum was opened, free amber-coloured fluid escaped, together with an enormously distended, purple loop of terminal ileum, about 18 in. long, which was seen to form part of the circumference of a large oval defect in the mesentery of the terminal ileum, about 4 × 2 in. across. Through this hole most of the small intestine had become herniated. Although the herniated bowel was not itself strangulated, the loop forming the margin of the defect was becoming so, from the occlusion of the marginal vessels lying in the free edge of the hole. All the bowel was still viable. The other viscera were normal; the cæcum and appendix lay in their normal situation.

After the herniated intestine had been replaced, the hole in the mesentery was sewn up with catgut on an eyeless needle, care being taken not to prick the vessels which lay in the rounded border of the defect. Owing to the distension of the ileum, it was not easy to close the abdomen, and might have been almost impossible without the aid of a muscle relaxant.

The baby was in a poor condition at the end of the operation, but responded to intravenous *N*/2 saline (20 ml./hr.) for the first two hours. This was followed by

Fig. 165.—Post mortem, showing sutured mesenteric defect.

Fig. 166.—Post mortem, showing extent of defect after removing catgut suture.

N/5 saline-dextrose (6 ml./hr.) for the next 18 hours. The same infusion was continued subcutaneously for another 6 hours, until, 24 hours after operation, oral feeds of 4 per cent dextrose in *N*/5 saline were given.

Meconium was passed after 12 hours, and bowel-sounds were heard. After 48 hours she was taking half-strength expressed breast milk in *N*/5 saline.

Penicillin (100,000 units b.d.) and streptomycin (60 mg. b.d.) had been given from the time of operation onwards. On the evening of June 22 she developed signs of pneumonia at the right base. The infecting organism was presumably insensitive to the antibiotics she had been given, so terramycin (100 mg. b.d.) was given instead. There was no response, and the baby died at 9.30 a.m. on June 23.

POST-MORTEM EXAMINATION (P.M. No. 292/59).—The cause of death was a severe bronchopneumonia in the right lung, with congestion and œdema of the left lung. The bowel was healthy. The repaired mesenteric defect was photographed (*Fig.* 165), and then the catgut was carefully unpicked to show the original size of the hole (*Fig.* 166). There were no other significant findings.

The culture of the pharyngeal swab which had been taken before death grew *Staph. pyogenes*, insensitive to penicillin, streptomycin, or terramycin, but sensitive to chloromycetin and oleandomycin.

DISCUSSION

Congenital defects in the mesentery of the small bowel are usually situated near the ileocæcal junction. They are only found in about 0·5 per cent of routine autopsies (Mitchell, 1899). Most of the reported cases have been in adults; a few have been in children over the age of 5 years; one other was in an infant. Internal herniæ are uncommon in children, but mesenteric defects are said by Gross (1953) to make up the largest single group amongst them. Swenson (1958) states that 10 per cent of all forms of intestinal obstruction in children are from this cause, but gives no evidence for such a high estimate.

At any age, the mortality has been very high, perhaps because a considerable length of bowel has had to be resected in many cases (Edwards, 1932; Williamson, 1933; Watson, 1937). It is curious that a congenital defect such as this should only give trouble in later years, and in this connexion it is interesting to read that Watson's patient, a 30-year-old negro, had had cramps after meals for nine years before the acute episode which brought him to laparotomy.

Other defects in the mesentery are described which have ragged edges and follow recent abdominal injury, or which have been preceded by some local intraperitoneal infection, but most of them have the features which were first carefully enumerated by Treves (1885): "The holes were round; they were situated in the mesentery of the terminal part of the ileum; their margins were distinct, being often thickened and opaque, and around a part of the margin it was not uncommon to find one of the terminal branches of the superior mesenteric artery. . . . In the fœtus it will often be observed that the ileocolic branch of the superior mesenteric artery circumscribes, by its anastomosis with the last of the intestinal arteries, an area on the mesentery of a well-rounded or oval shape. . . an area so differentiated from the surrounding mesentery I have seen in fœtuses of six and seven inches in length, but although a common, it is not a constant condition. . . . It will be seen that this area has the precise situation, the outline, and the dimensions of the mysterious mesenteric hole; and by the atrophy of the peritoneum occupying the district such a hole would be formed."

Not only is the peculiar relationship of the vascular loop between mesenteric and ileocolic arteries of significance in the origin of these defects, it is also a hazard of the operation. If the neck of the hernial opening is tight, there is no safe place where it can be divided, and Bailey (1958) recommends emptying the strangulated bowel with an aspirating needle to effect reduction of the hernia. It may also be impossible to suture together the edges of the foramen without injuring one of these vessels; Baty (1938) noted this feature in his case, and judged it wiser to leave the hole open.

That this infant should survive the perils of internal strangulation on the second day of life, only to perish of a pneumonia on the fifth, demands some explanation. Many factors may be indicted: she was puny from birth; at the outset she may have inhaled some vomit; the duration of the operation—half an hour—was probably excessive for one so little; the endotracheal tube and the inhalation anæsthetic may have been injurious; the right paramedian incision—long in proportion to the small abdomen—must have limited the movements of the right side of the chest; finally, the pathogenic organism was insensitive to the prophylactic combination of antibiotics used for choice, as well as to the second line of defence, terramycin. It is always possible to give too much intravenous fluids in such a case but the quantities, as set out in detail above, were jealously regulated, and do not even in retrospect seem culpable.

SUMMARY

A case of intestinal obstruction in a 2-day-old baby was found to be caused by internal herniation through a large defect in the mesentery. Death took place from pneumonia four days after operation.

It is a pleasure to thank Professor Dix, Dr. Anthony Jackson, and Mr. Leonard Easton for their help with this paper, and for their permission to publish this case, which was in their care; Drs. W. H. Marshall and A. F. Hayward for their help; and Messrs. John King and R. F. Ruddick for the photographs.

REFERENCES

BAILEY, HAMILTON (1958), *Emergency Surgery*, 7th ed., 432. Bristol: John Wright.
BATY, J. A. (1938), *Brit. med. J.*, **1**, 671.
EDWARDS, C. R. (1932), *J. Amer. med. Ass.*, **99**, 278.
GROSS, R. E. (1953), *The Surgery of Infancy and Childhood*, 447. Philadelphia: W. B. Saunders.
HANSMANN, G. H., and MORTON, S. A. (1939), *Arch. Surg., Chicago*, **39**, 973.
LONG, E. R. (1927), *Trans. Chicago Path. Soc.*, **12**, 335.
MITCHELL, L. J. (1899), *Ann. Surg.*, **30**, 505.
SWENSON, O. (1958), *Pediatric Surgery*, 327. New York: Appleton-Century-Crofts.
TREVES, F. (1885), *Brit. med. J.*, **1**, 470.
WATSON, J. R. (1937), *Ann. Surg.*, **106**, 1097.
WILLIAMSON, J. C. F. L. (1933), *Brit. J. Surg.*, **20**, 684.

FISTULA FORMATION BETWEEN THE DUODENUM AND COLON

A REPORT OF THREE CASES AND A REVIEW OF THE CONDITION

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A CASE of fistula formation between the duodenum and colon was described in 1862 by Haldane; in this case a cæcocolic fistula was present as well, and both were said to have a malignant origin. In the following year Sanderson (1863) reported a duodenocolic fistula opening into the transverse colon from a duodenal pouch. This case was thought to be benign. Malignant fistulæ are more frequently seen than benign, and the greater number are due to carcinoma of the colon in the region of the hepatic flexure. Infiltration of the second part of the duodenum by the growth and ultimately erosion and fistula formation occur.

Pathology.—Cases of malignant fistulæ due to carcinoma of the colon have been reported by Mindline and Rosenheim (1935), Saleh (1935), Carnot and Caroli (1936), Hubeny and Delano (1940), Feldman (1942), Linton (1944), Calmenson and Black (1947), Hall Drake and Goodwin (1948), Halligan (1950), and Baker (1952). Hershenson and Kirsner (1951) recorded that 32 cases, benign and malignant, had been reported, 19 resulting from carcinoma of the colon.

Benign fistulæ show a considerable aetiological variety. The commonest cause is a ruptured duodenal ulcer (McPeak, 1940; McClinton, 1944; Railton, 1948; Rife, 1951; Kroch, 1950; and Lyons, 1951). Ogilvie (1950) reported 2 cases following tuberculous lymphadenitis. Two cases of fistula formation as a result of ulcerative colitis are described by Ransom (1951). Ormandy and Bargen (1939) have also recorded a case in which gastrocolic and duodenocolic fistulæ were associated with ulcerative colitis.

The present paper includes a case in which a carcinoma of the gall-bladder eroded into the duodenum and hepatic flexure, resulting in a duodenocolic fistula. This appears to be the only case reported with such an aetiology.

Clinical Picture.—The symptoms are likely to be those of the underlying lesion, with the characteristic results of fistula formation superimposed. If the condition is benign, then the symptoms may be due almost entirely to the fistula. Loss of weight and diarrhœa and vomiting are marked features. The stools may contain undigested food, and, as the condition advances, fæcal vomiting often occurs, which should give rise to suspicion of such a lesion. In a short period the patient becomes emaciated and dehydrated and a severe macrocytic anæmia is common. Hypoproteinæmia, with reversal of the albumin-globulin ratio and œdema, may occur. Avitaminosis and electrolyte disturbances, with their characteristic manifestations, may occur and be severe (Halligan, 1950). The calcium-phosphate ratio may be upset and the case may present as one of idiopathic steatorrhœa (Mindline and Rosenheim, 1935). Clubbing of the fingers may occur. The general deterioration and loss of weight can be extremely rapid. It should be noted, however, that many of the symptoms described may be the result of advanced malignant disease alone.

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CASE REPORTS

Case 1.—Male, aged 77 years, a retired game-keeper. Admitted to Kettering General Hospital on June 3, 1958, complaining of severe diarrhœa, vomiting, and upper abdominal pain for three days.

HISTORY.—A very fit, active man until about two years previously, when he started to develop pain in the upper abdomen a quarter to half an hour after meals. On increasingly frequent occasions this was associated with

FIG. 167.—*Case* 1. *Barium Meal.* The fundus only of the stomach is filled with barium, the antrum and body being air filled. No lesion is seen in the stomach. The cæcum and ascending colon are filled with barium and the narrow fistulous communication between colon and duodenal loop is well demonstrated. The small intestine is slightly dilated.

nausea and, very occasionally, vomiting which eased the pain. Not helped by alkalis, but was considerably helped by a sloppy milky diet, which he had continued to take for the past two years. During the last two years he had episodes of complete freedom from these symptoms, but noticed that in the past year he had been getting weaker, and had to give up his part-time job. In the past year he had suffered somewhat with flatulence and found that this was worse with the sloppy diet. An attack of pleurisy occurred in December, 1957, and subsequently diarrhœa and vomiting developed. Vomit, which had previously been greenish in colour, was now occasionally dark brown, with a very foul odour, but he did not notice any regurgitated food, although vomiting occurred quite quickly after meals. For the past four months the diarrhœa had been troublesome, with 5-6 loose motions a day, and in the last three months he had noticed that his weight had dropped from a normal 11 st. to about 7 st. His general health was poor and he had become progressively weaker. He had never passed blood or slime per

rectum. In the last ten years he had noticed some nocturia once or twice and urgency. He had never been overseas and had never previously been in hospital.

Past History.—Typhoid at the age of 25 years. Ill for one year.

ON EXAMINATION.—A very pale, dehydrated, ill man. Temperature 97° F., pulse-rate 90, blood-pressure 140/60. No ankle or sacral œdema. Tender mass in the epigastrium just to the left of the midline. Rectal examination: no abnormality detected.

Investigations.—

Blood: Hb/47 per cent, 6·9 g. per cent; R.B.C., 2,150,000 per c.mm.; W.B.C., 6300 per c.mm. Differential count: normal. Wintrobe E.S.R., 6 mm./hr.

Serum-proteins: Total, 4·3 g. per cent; albumin, 1·8 g. per cent; globulin, 2·5 g. per cent. A/G ratio, 0·72 : 1.

Blood-urea: 40 mg. per cent. Alkaline phosphatase, 6·0 units per cent. Serum electrolytes: normal.

Barium Meal (June 9).—The stomach was of the cup-and-spill variety and held barium for a considerable period in the fundus. The body and antrum were gas-filled for the most part, and not clearly demonstrated. There was some dilatation of the small intestine. Early in the examination a considerable quantity of barium filled what appeared to be the ascending colon and cæcum. A small track was seen running from this area to the duodenum. (*Fig*. 167.)

Conclusion: "The appearance suggested that there was a fistulous communication between the ascending colon and either the duodenum or antrum of the stomach, more likely the duodenum." The cause of this condition is usually a carcinoma in the region of the hepatic flexure.

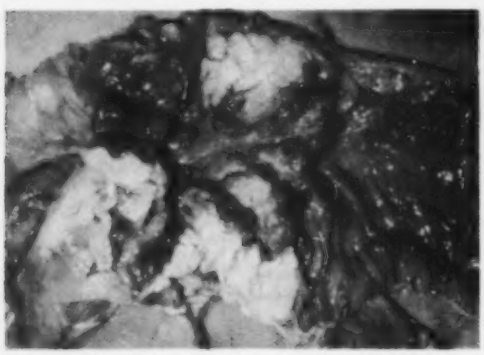

FIG. 168.—*Case* 1. The resected portion of colon (hepatic flexure) is opened out and displayed, showing the growth almost filling the lumen and the fistula demonstrated by the probe. At the upper end of the probe is some duodenal mucosa adherent to the growth.

He was transfused on June 9 with 4 pints of blood, and his hæmoglobin two days later was 72 per cent. He was given two low-pressure large-bowel wash-outs following a high enema, with good result, prior to laparotomy.

AT OPERATION (Mr. T. H. Cullen).—On June 11, under general anæsthesia, the abdomen was opened by an upper midline incision, and a large carcinoma of the hepatic flexure was found to be closely adherent to the medial aspect of the second part of the duodenum. The ascending and transverse colon were mobilized and the ileum was divided some 18 in. from the ileocæcal junction. After mobilization it was seen that the duodenal wall was invaded by the tumour and the colon and duodenum were connected by a fistulous tract. Right hemicolectomy was performed and that part of the duodenal wall involved in the growth was excised in continuity with the colon. The duodenal wall was repaired transversely and an end-to-end ileotransverse anastomosis was performed. No metastases were seen in the liver or peritoneal cavity.

Post-operatively his progress was very satisfactory and his hæmoglobin rose to 85 per cent on June 30. During this post-operative period he developed acute retention due to a benign hypertrophy of the prostate and a temporary Riches suprapubic cystostomy was performed. He was discharged from hospital on July 23, some four and a half weeks after admission. On discharge he was eating a normal diet, with no abdominal pain or vomiting and passing normal, well-formed motions once a day.

When last seen in October he had gained 1½ st. in weight and was passing urine normally per urethram, no prostatectomy being necessary. Histological section showed a well-differentiated adenocarcinoma of the colon invading all layers of the colon and the attached part of the duodenum. A fistula was present between the colon and duodenum through the tumour (*see Fig*. 168).

FIG. 169.—*Case* 2. *Barium Meal*. As in *Fig*. 168, barium fills the fundus only, the remainder of the stomach being gas filled. A narrow track runs from the duodenal loop to the ascending colon, which is not well filled and has many diverticula.

Case 2.—Male, aged 70 years. Admitted to Northampton General Hospital on Aug. 7, 1958 complaining of vomiting, diarrhœa, and abdominal pain for about 6 weeks. In addition there had been considerable weight-loss. The diarrhœa was violent and occurred very shortly after eating or drinking.

ON EXAMINATION.—Anæmic, dehydrated, and very weak. No palpable masses in the abdomen. There was œdema of the right ankle and of the stump of the left leg, which had previously been amputated.

Special Investigation.—

Hæmatology: Hb/46 per cent; W.B.C., 2900 per c.mm. Differential count: normal. E.S.R., 28 mm./1 hr. (Wintrobe).

Occult blood: Positive.

Plasma-proteins: Total, 4·6 g. per cent; albumin, 1·8 g. per cent; globulin, 2·8 g. per cent. A/G ratio, 0·6 : 1.

Blood-urea: 32 mg. per cent.

Barium Enema Report.—"There is a fistula between the large gut and the proximal part of the alimentary

tract, so that the stomach and duodenum were filled on barium examination. Numerous diverticula of the distal half of the colon are present. The site of the fistula is not determined, but probably is at the ascending colon to proximal jejunum. A barium meal might help. The fistula is probably secondary to a carcinoma of the colon, although a perforated diverticulum is not entirely excluded."

Barium Meal (*Fig.* 169).—"No abnormality of the œsophagus, stomach, or duodenal cap. There was marked tenderness over the duodenal loop. The small-gut-large-gut fistula is probably from the second and third parts of the duodenum to the transverse colon. At first barium did not pass the duodenal loop easily. Food was given and at three hours the stomach was empty. Diluted barium was shown in the colon. Concentrated barium remained in the small gut."

AT OPERATION (Mr. E. E. T. Taylor).—Under a general anæsthetic the abdomen was opened by a right paramedian incision. A mass was present in the hepatic flexure of the colon and a fistula from the third part of the duodenum to the hepatic flexure. No signs of metastases in the peritoneal cavity. A right hemicolectomy and ileotransverse anastomosis was performed. The duodenal fistula was repaired. The wound was closed with drainage to the site of the anastomosis.

PATHOLOGICAL REPORT (Sept. 14).—Specimen: Colon, 25 cm. In the middle of the specimen there is an ulcerated tumour 4 cm. across, which is completely encircling the gut and penetrating through all coats. A small portion of the duodenum is adherent to the base of the tumour and there is a fistula through this.

Microscopic Examination.—An infiltrating adenocarcinomatous growth showing lymphatic involvement.

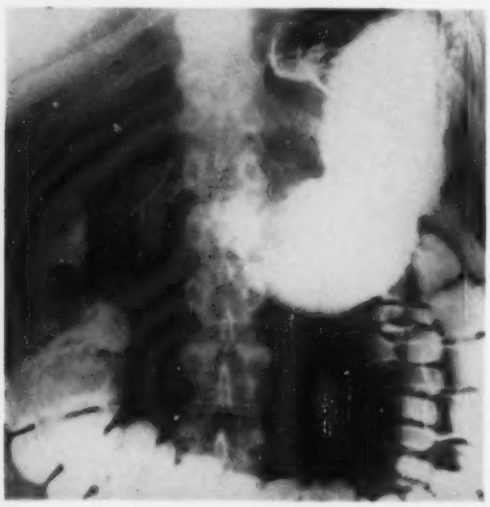

FIG. 170.—*Case* 3. *Barium Meal.* In this case the stomach is well filled with barium and there is marked irregularity of the first and second parts of the duodenum. The fistula is shown communicating with the hepatic flexure and the colon has become almost completely filled with barium, giving the appearance of a barium enema rather than a meal.

Case 3.—Female, aged 67 years. Admitted April 17, 1954. (The radiological features of this case were reported by Medhurst (1956).)

HISTORY.—This patient was admitted as an emergency to Mayday Hospital, Croydon, under Mr. C. F. Swinton, with vomiting and diarrhœa of five days' duration. There had been melæna for two days and general malaise for about a week. She complained of pain in the right side of the abdomen which radiated to the inferior angle of the scapula.

Past History.—A history of gall-stones for about 30 years and in October, 1952, an acute empyema of the gall-bladder was diagnosed. A cholecystostomy was carried out. In February, 1953, a cholecystoduodenostomy was performed.

CLINICAL EXAMINATION.—A hard, tender mass was noted in the right hypochondrium which moved on respiration. Some tenderness was present in the right iliac fossa, but no rigidity was present. The bowel-sounds were normal and the tongue furred.

SPECIAL EXAMINATION.—Hb 46 per cent; W.B.C., 16,000 per c.mm.; blood-urea, 37 mg. per cent. Fæces: no pathogens isolated on culture. Occult blood, positive.

Barium-meal Report.—"Œsophagus—normal. Barium entered the stomach and passed without difficulty to the duodenal cap. Beyond this point there was irregularity of the lumen and no further passage of the barium was noted. Radiographs (*Fig.* 170), taken an hour later, showed that barium passed directly from the duodenum to the ascending colon, and in this short time the whole colon was filled with barium. The appearance was consistent with a fistula between the colon and the duodenum. Comment: The cause is probably the result of operative treatment or a neoplastic lesion involving the duodenum and hepatic flexure."

Barium Enema Report.—"It was not possible to get the barium beyond the hepatic flexure at the place where, in the barium meal, an apparent duodenocolic fistula was present. By superimposing the barium enema films the anatomy is made clear." No barium passed from the colon via the duodenum into the stomach at the enema examination.

OPERATION (Mr. C. F. Swinton).—Right Kocher incision. A hard, irregular, nodular mass was present involving the gall-bladder, liver, and hepatic flexure. The case was inoperable. The wound was closed after a gland from the omentum was taken for biopsy.

PATHOLOGICAL REPORT.—A fairly well-differentiated adenocarcinoma.

CONCLUSION.—A duodenocolic fistula occurring as a result of a gall-bladder carcinoma which had involved the liver, hepatic flexure, and duodenum.

DISCUSSION

All the patients were elderly, the youngest being 67 years and the oldest 77 years of age. All suffered from advanced malignant disease, 2 from carcinoma of the colon in the hepatic flexure region, and the third from a gall-bladder tumour which had spread to the adjacent viscera, involving the liver, duodenum, and colon. Right-sided colonic carcinoma is the commonest cause of duodenocolic fistula, and it is not surprising that 2 of the 3 cases reported had this origin. The third case of fistula formation following a gall-bladder neoplasm must be extremely rare and no mention was found of a similar case in a fairly extensive survey of the literature. There was a history in this case of gall-stones for about 30 years and an empyema of the gall-bladder.

All 3 patients were in poor condition and suffered from vomiting and diarrhœa at a late stage in their illness. All had lost a considerable amount of weight. One patient, describing his diarrhœa, stated that "it suddenly shoots out after eating or drinking—about one pint at a time".

In all cases there was a strong suspicion of malignant disease and barium examination demonstrated the presence of fistula formation.

Radiology.—In the demonstration by barium enema, supine, prone, and post-evacuation radiographs

are required. There is frequently an obstruction to the flow of barium, usually in the hepatic flexure or right part of the transverse colon, together with constriction or filling defect. The fistula may be clearly seen and a sudden flooding of the small intestine may occur. Barium meal examination is usually considered less successful in demonstrating the fistula. There may be delay in the passage of barium through the duodenal loop, and follow-through studies may demonstrate the fistulous communication.

It is generally considered that examination by barium enema is the most satisfactory method of fistula demonstration. In the 3 cases examined, however, this was not found to be the case. In the first case the fistula was shown clearly by barium meal and no further examination was carried out owing to the frail condition of the patient. In the second case, the fistula was shown in both barium meal and enema. In the third case, a good demonstration was made by meal but a barium enema did not show the lesion.

It is reasonable to assume that if a fistula is suspected, both barium meal and enema should be carried out, as one may give positive, and the other negative, results. In the 3 cases described the meal was the more efficient method of examination—this is contrary to the general findings in the literature.

Treatment.—This depends to a large extent on whether the lesion is benign or malignant. *Benign*: Simple excision of the fistulous tract and closure may be sufficient, but involvement of the colon or duodenum by infection or stricture may necessitate radical methods such as hemicolectomy. *Malignant*: The treatment of fistulous cases due to a primary malignant growth, usually of the colon, is essentially that of treating the tumour. Invariably when fistula formation occurs, the growth is advanced and secondary deposits are often already present, and so the treatment becomes one of palliation and relief of troublesome symptoms such as vomiting and diarrhœa. Gastroenterostomy and cæcostomy may be the only adequate methods of making life tolerable. The primary growth is usually situated in the hepatic flexure of the colon and, in operable cases, right hemicolectomy and ileotransverse anastomosis are performed. In addition the fistulous tract and that part of the duodenal wall involved in the growth are excised and a simple closure of the duodenal wall is performed. This was the procedure in *Cases* 1 and 2. Thus operative treatment is essentially that of a right hemicolectomy with supportive therapy in the form of blood and plasma transfusions. Avitaminosis and steatorrhœa caused by the fistula are quickly corrected when a normal diet is again started.

SUMMARY

Three cases of duodenocolic fistula are described, 2 occurring as a result of carcinoma of the hepatic flexure region, and the third as a complication of gall-bladder carcinoma, with massive involvement of the adjacent viscera.

Laparotomy was performed in all cases and successful resection was carried out in 2 of the 3 cases. In the last case the spread was too great for successful resection to be attempted.

Radiological demonstration of the lesion was more satisfactory with a barium meal than with a barium enema, which is unusual and contrary to most writers' experience.

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REFERENCES

BAKER, H. K. (1952), *J. Mo. med. Ass.*, **49**, 49.
CALMENSON, M., and BLACK, B. M. (1947), *Surgery*, **21**, 476.
CARNOT, P., and CAROLI, J. (1936), *Bull. Soc. méd. Hôp. Paris*, **52**, 234.
DRAKE, E. P. HALL, and GOODWIN, J. F. (1948), *Brit. J. Surg.*, **36**, 204.
FELDMAN, M. (1942), *Amer. J. dig. Dis.*, **9**, 195.
HALDANE, D. R. (1862), *Edinb. med. J.*, **7**, 624.
HALLIGAN, E. J. (1950), *Amer. J. Proctol.*, No. 1, March.
HERSHENSON, L. M., and KIRSNER, J. B. (1951), *Gastroenterology*, **19**, 864.
HUBENY, M. J., and DELANO, P. J. (1940), *Amer. J. Roentgenol.*, **43**, 198.
KROCH, F. (1950), *see* OGILVIE. Discussion of paper given by Ogilvie.
LINTON, R. R. (1944), *Arch. Surg., Chicago*, **48**, 197.
LYONS, C. (1951), quoted by OLSEN, personal communication.
MCCLINTON, J. B. (1944), *Canad. med. Ass. J.*, **2**, 434.
MCPEAK, C. N. (1940), *Radiology*, **34**, 343.
MEDHURST, G. A. (1956), *Brit. J. Radiol.*, **29**, 381.
MINDLINE, J., and ROSENHEIM, M. L. (1935), *Lancet*, **2**, 764.
OGILVIE, Sir H. (1950), *Ann. Surg.*, **131**, 899.
ORMANDY, L., and BARGEN, J. A. (1939), *Proc. Mayo Clin.*, **14**, 550.
RAILTON, S. V. (1948), *Canad. med. Ass. J.*, **59**, 267.
RANSOM, H. K. (1951), quoted by RIFE.
RIFE, C. S. (1951), *Arch. Surg., Chicago*, **62**, 876.
SALEH, A. H. (1935), *Lancet*, **2**, 1117.
SANDERSON (1863), *Trans. Path. Soc. Lond.*, **14**, 173.

PEPTIC ULCERATION IN INFANCY

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Gastric and duodenal ulceration in infancy is rare; recognition of this condition is first credited to Siebold (1825), who described the post-mortem findings in a neonate in whom perforation of a duodenal ulcer had occurred. Shortly after, Cruveilhier (1829) described 3 cases of gastric ulceration in children below the age of 4 months. Since these initial reports intermittent communications concerning peptic ulceration in childhood have appeared in the literature. Bird, Limper, and Mayer (1941), in an extensive review of the literature, collected all the recorded cases of this condition. They found that 119 cases of peptic ulceration had been reported in children below 15 years of age in whom varied types of operation had been performed, and an additional 124 cases in which treatment had been conservative. Out of this total of over 200 cases, 96 patients, i.e., 39 per cent, were children below the age of 1 year, and of this group only 15 were submitted to surgery, the indications for operation being pyloric obstruction in 4, perforation in 8, and bleeding in 3 cases. In this small group of 96 patients the overall mortality was 60 per cent and the mortality for gastric resection 100 per cent.

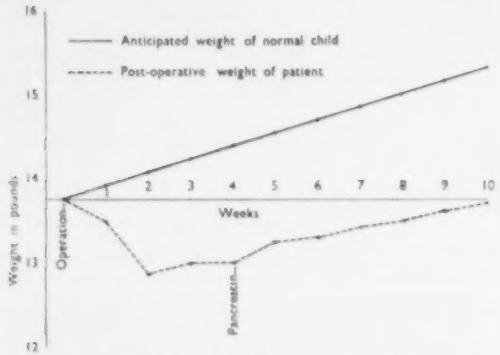

Fig. 171.—Weight-gain of infant subjected to partial gastrectomy as compared with weight-gain of normal infant.

Examination of these data shows that in infants under two weeks of age duodenal ulceration is twice as common as gastric ulceration; bleeding, perforation, or both, are the common methods of presentation and characteristically the onset of symptoms is precipitous. In all the cases in this group recorded by Bird and others (1941) ulceration was associated with either an intracranial lesion or infection. No mention is made by these authors of the association of duodenal ulceration with suprarenal hæmorrhage, although these two factors are probably linked together by a common factor of infection (Gunson, 1914).

In older infants duodenal ulceration as compared with gastric ulceration was even commoner and, whilst perforation and bleeding were frequent, prodromal symptoms such as refusal of feeds or vomiting occurred, drawing attention to the upper gastro-intestinal tract prior to the occurrence of the catastrophe.

In 1942 Guthrie, after finding at autopsy a duodenal ulcer in a child of 10 weeks who had died following multiple rectal hæmorrhages, reviewed the records of the Royal Hospital for Sick Children in Glasgow. Out of 6059 autopsies performed between the years 1914 and 1941, 9 cases of peptic ulceration were found. Such evidence confirms the clinical impression that duodenal ulceration in infancy is rare and that it is a disease carrying a high mortality. Furthermore, even if laparotomy is performed the condition may be overlooked, as in the case reported by Franklin (1942).

The following case of duodenal ulceration in infancy is therefore recorded, since it illustrates the difficulties associated with the pre-operative diagnosis and some aspects of post-gastrectomy physiology in the infant.

CASE REPORT

The patient, a female infant aged 5 months, was admitted to the General Infirmary at Leeds under the care of Professor W. S. Craig on Jan. 26, 1959.

History.—The infant was well until one week prior to admission, at which time the mother noticed the child had a running nose. After three days the baby developed frequency of micturition and a napkin rash. Twenty-four hours prior to admission the baby started screaming attacks and at the same time the mother noted grunting respiration.

Physical Examination.—A well-nourished child, weighing 14 lb.

Respiratory System.—The child was not cyanosed but its respiratory rate was 60/min., and dullness to percussion was detected over the right base.

Special Investigations.—

Total white blood-count: 13,200; polymorphonuclear leucocytes 92 per cent.

Radiography of chest: An opacity situated over the right lower chest suggested the presence of pneumonic consolidation or an empyema.

Aspiration of chest: A small quantity (2 ml.) of pus was obtained from which *Staph. aureus* was cultured. The organism was sensitive to penicillin (2 units/100 ml.), erythromycin, and novobiocin (2 μg./100 ml.).

Treatment.—Following aspiration of the chest 100,000 units of penicillin were instilled into the pleural cavity and intramuscular penicillin, 150,000 units b.d., was commenced.

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Once again the child's general condition improved until Feb. 9, on which date two large stools composed of bright-red blood were passed; the melæna continued intermittently throughout the next 48 hours and in spite of apparently adequate transfusion the hæmoglobin fell to 44 per cent.

In view of the persistent melæna laparotomy was performed on Feb. 11.

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Procedure: A Polya type-II gastrectomy was performed. The gastroduodenal artery was ligated at its origin from the hepatic and the duodenal stump was infolded. An antecolic afferent loop to greater curve gastrojejunostomy was performed removing approximately one-half of the stomach. The anastomosis was performed throughout with 00 chromic catgut without clamps.

POST-OPERATIVE COURSE.—Intermittent gastric suction was employed for 24 hours, after which, following a loose bowel action, oral feeding was started with 20 oz. of half-cream National Dried Milk. The weight of the child throughout the first ten post-operative weeks is shown in *Fig.* 171, together with the anticipated gain in weight of a normal infant. Four weeks after operation pancreatin (Paines & Byrne), gr. 125 a day, was given in divided doses intimately mixed with each feed.

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TIME IN HR.	09.00	10.00	11.00	12.00	13.00	14.00	15.00	16.00	17.00	18.00	19.00	20.00
Test 1: No milk taken by mouth	4·9	5·0	5·0	5·1	5·0	5·0	4·9	4·9	4·9	4·9	4·8	4·9
Test 2: Normal 3-hourly feeding	3·7	5·9	4·7	6·3	4·5	—	4·6	5·3	7·0	6·5	7·3	7·4
Test 3: *p*H of adult gastric remnant	3·2	4·3	3·5	3·5	3·7	3·7	3·7	3·3	3·7	3·8	4·0	4·2

TIME IN HR.	21.00	22.00	23.00	24.00	01.00	02.00	03.00	04.00	05.00	06.00	07.00	08.00
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Table II.—CHANGES IN BLOOD-SUGAR AND SERUM-POTASSIUM AFTER INGESTION OF GLUCOSE

	CONTROL	40 MIN.	70 MIN.	100 MIN.	120 MIN.
Blood-sugar	120 mg. per cent	120 mg. per cent	90 mg. per cent	70 mg. per cent	90 mg. per cent
Serum K	4·8 mEq./l.	4·2 mEq./l.	4·1 mEq./l.	4·1 mEq./l.	4·1 mEq./l.

4. *Measurement of Fæcal Fat Excretion.*—The fæcal fat was estimated on a dietary fat intake of 12·9 g./day, and secondly after increasing the intake to 16 g. In addition, during the latter period gr. 15 of pancreatin were added to each feed.

The fæcal fat without pancreatin expressed as a percentage of total fat intake varied from 25 per cent to 75 per cent in three 2-day samples; after the administration of pancreatin the fæcal fat absorption varied from 46 per cent to 57·5 per cent.

DISCUSSION

As previously stated, Bird and others (1941) divided the recorded cases of peptic ulceration in childhood into two groups dependent upon age. The lesion described in this communication would appear to belong to their first type—that is, an ulcer developing in the presence of infection and/or an intracranial lesion. However, in the case reported here the age of

PEPTIC ULCERATION IN INFANCY

By G. LEWIS, D. PRATT, and F. G. SMIDDY
DEPARTMENTS OF PÆDIATRICS AND SURGERY, GENERAL INFIRMARY AT LEEDS

GASTRIC and duodenal ulceration in infancy is rare; recognition of this condition is first credited to Siebold (1825), who described the post-mortem findings in a neonate in whom perforation of a duodenal ulcer had occurred. Shortly after, Cruveilhier (1829) described 3 cases of gastric ulceration in children below the age of 4 months. Since these initial reports intermittent communications concerning peptic ulceration in childhood have appeared in the literature. Bird, Limper, and Mayer (1941), in an extensive review of the literature, collected all the recorded cases of this condition. They found that 119 cases of peptic ulceration had been reported in children below 15 years of age in whom varied types of operation had been performed, and an additional 124 cases in which treatment had been conservative. Out of this total of over 200 cases, 96 patients, i.e., 39 per cent, were children below the age of 1 year, and of this group only 15 were submitted to surgery, the indications for operation being pyloric obstruction in 4, perforation in 8, and bleeding in 3 cases. In this small group of 96 patients the overall mortality was 60 per cent and the mortality for gastric resection 100 per cent.

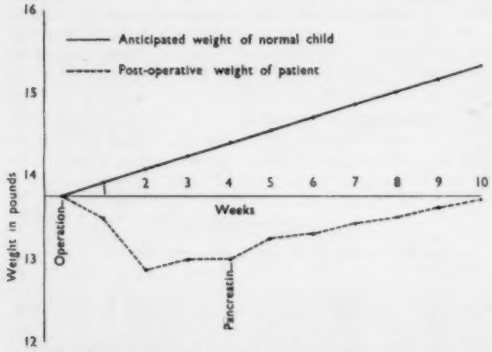

FIG. 171.—Weight-gain of infant subjected to partial gastrectomy as compared with weight-gain of normal infant.

Examination of these data shows that in infants under two weeks of age duodenal ulceration is twice as common as gastric ulceration; bleeding, perforation, or both, are the common methods of presentation and characteristically the onset of symptoms is precipitous. In all the cases in this group recorded by Bird and others (1941) ulceration was associated with either an intracranial lesion or infection. No mention is made by these authors of the association of duodenal ulceration with suprarenal hæmorrhage, although these two factors are probably linked together by a common factor of infection (Gunson, 1914).

In older infants duodenal ulceration as compared with gastric ulceration was even commoner and, whilst perforation and bleeding were frequent, prodromal symptoms such as refusal of feeds or vomiting occurred, drawing attention to the upper gastro-intestinal tract prior to the occurrence of the catastrophe.

In 1942 Guthrie, after finding at autopsy a duodenal ulcer in a child of 10 weeks who had died following multiple rectal hæmorrhages, reviewed the records of the Royal Hospital for Sick Children in Glasgow. Out of 6059 autopsies performed between the years 1914 and 1941, 9 cases of peptic ulceration were found. Such evidence confirms the clinical impression that duodenal ulceration in infancy is rare and that it is a disease carrying a high mortality. Furthermore, even if laparotomy is performed the condition may be overlooked, as in the case reported by Franklin (1942).

The following case of duodenal ulceration in infancy is therefore recorded, since it illustrates the difficulties associated with the pre-operative diagnosis and some aspects of post-gastrectomy physiology in the infant.

CASE REPORT

The patient, a female infant aged 5 months, was admitted to the General Infirmary at Leeds under the care of Professor W. S. Craig on Jan. 26, 1959.

HISTORY.—The infant was well until one week prior to admission, at which time the mother noticed the child had a running nose. After three days the baby developed frequency of micturition and a napkin rash. Twenty-four hours prior to admission the baby started screaming attacks and at the same time the mother noted grunting respiration.

PHYSICAL EXAMINATION.—A well-nourished child, weighing 14 lb.

Respiratory System.—The child was not cyanosed but its respiratory rate was 60/min., and dullness to percussion was detected over the right base.

SPECIAL INVESTIGATIONS.—

Total white blood-count: 13,200; polymorphonuclear leucocytes 92 per cent.

Radiography of chest: An opacity situated over the right lower chest suggested the presence of pneumonic consolidation or an empyema.

Aspiration of chest: A small quantity (2 ml.) of pus was obtained from which *Staph. aureus* was cultured. The organism was sensitive to penicillin (2 units/100 ml.), erythromycin, and novobiocin (2 μg./100 ml.).

TREATMENT.—Following aspiration of the chest 100,000 units of penicillin were instilled into the pleural cavity and intramuscular penicillin, 150,000 units b.d., was commenced.

PROGRESS.—The child's general condition gradually improved on this régime, although a blood culture taken five days after admission grew *Staph. aureus*.

On Feb. 1, however, the child's condition suddenly deteriorated, profuse diarrhœa associated with a high temperature developed, followed some hours later by cyanosis and impalpable peripheral pulses. Following resuscitation with intravenous fluids it became obvious clinically that the child had sustained cerebral damage, since there was marked neck rigidity and inability to feed from a bottle. In addition it was noted that the great toe on the left foot was blue and cold. Cultures of the stools during the period of diarrhœa failed to reveal any pathogenic organisms.

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the child is appreciably greater, 20 weeks at onset, as compared with 2 weeks, and in addition both infection and cerebral factors were in operation. One may assume that peptic ulceration developed in this child at the time of the cerebral catastrophe, an assumption supported by the coincident development of digital thromboses in the left foot.

Thus the onset of bleeding from the gastro-intestinal tract in an infant at any age, particularly in the presence of other areas of vascular injury, should alert one to the possibility of peptic ulceration. That the stools in this child were composed of almost fresh blood led to the erroneous belief that the source of hæmorrhage lay low down in the gastro-intestinal tract and a pre-operative diagnosis of a bleeding Meckel's diverticulum was made.

In the adult the presence of peptic ulceration in a patient bleeding from the gastro-intestinal tract can be readily confirmed by a barium meal performed during the course of the hæmorrhage (Cantwell, 1959), and from the pathological findings at operation in this child it seems probable that this technique would have confirmed the presence of duodenal ulceration in this case. Barium or lipiodol meals in the adult have proved to be quite free of danger and are also helpful in assessing the probable response of the patient to medical treatment.

The operative findings in this child confirm the impression gained from the literature that peptic ulceration may be overlooked even when laparotomy is performed, and in this patient the importance of open inspection of the duodenum is clearly demonstrated. It would seem logical that if this procedure had been negative gastrotomy should have followed closure of the duodenum.

Reference to *Fig.* 171 shows that the weight of the child did not increase following operation for a period of 4 weeks, although the loss was considerably less than the 20 per cent body-weight loss which Smith (1951) states can be tolerated by the infant without harm. Following the finding of greatly diminished fat absorption pancreatin was added to the feeds, a procedure recommended by Harris, Norman, and Payne (1955) in the treatment of pancreatic insufficiency and well documented in the treatment of adult post-gastrectomy steatorrhœa.

The weight increase which followed the use of pancreatin would suggest that this therapy was effective, even though examination of the fæcal fat showed no consistent increase in fat absorption. Unfortunately nitrogen balances were not performed, but it is difficult to believe, especially in view of the work of Harris and others (1955), that an increase in protein utilization could have occurred in the absence of an increased fat absorption.

Examination of *Table I* shows that the pattern of acidity in the post-gastrectomy remnant in the infant is considerably less than in the adult, and this difference in pattern is particularly obvious during the nocturnal period. Smiddy (1957) suggested that in the post-gastrectomy remnant nocturnal acidity remained high owing to the continuing influence of vagal stimuli, but the results described here suggest that nocturnal vagal stimulation of gastric secretion in the infant is of considerably less significance than in the adult. The low acidity in the gastric pouch is probably not due to the absence of acid secretion, since Miller (1942) established that in all but 12 per cent of normal breast-fed infants acid secretion occurred.

'Dumping' as a recognizable syndrome could not occur in the infant, although the mechanical factors necessary for its production are present. It was for this reason that glucose meals were given; the results show that after glucose meals the blood-sugar rapidly rises, to be followed by a relative hypoglycæmia, these changes being associated with variations in the serum-potassium. However, in spite of the changes in the potassium level no changes in the electrocardiogram were observed.

Although this infant has satisfactorily recovered from gastrectomy and appears to be gaining weight by relatively normal increments, the presence of the continued and apparently uncontrollable steatorrhœa suggests that this operation is perhaps too disturbing of gastro-intestinal physiology in the infant to be acceptable. Since it can be argued that the factors determining the onset of ulceration, i.e., infection and/or vascular damage, are self-limiting, this suggests that ligation of the gastroduodenal artery above and below its transit through the ulcer crater would have been a more acceptable method of treatment, this to be supplemented by active medical measures to ensure complete anacidity in the stomach.

We gratefully acknowledge the help given by Professor W. S. Craig in the preparation of this manuscript.

REFERENCES

BIRD, C. E., LIMPER, M. A., and MAYER, J. M. (1941), *Ann. Surg.*, **114**, 526.
CANTWELL, D. (1959), personal communication.
CRUVEILHIER, J. (1829), *Anatomie pathologique du corps humain*. Paris: J. B. Ballière.
FRANKLIN, A. W. (1942), *Arch. Dis. Childh.*, **17**, 95.
GUNSON, E. B. (1914), *Proc. R. Soc. Med.*, **8**, 54.
GUTHRIE, K. (1942), *Arch. Dis. Childh.*, **17**, 82.
HARRIS, R., NORMAN, A. P., and PAYNE, W. W. (1955), *Ibid.*, **30**, 424.
MILLER, R. A. (1942), *Ibid.*, **17**, 198.
SIEBOLD, H. (1825), *J. Geburtsh.*, **5**, 1.
SMIDDY, F. G. (1957), *Gastroenterology*, **32**, 1066.
SMITH, C. A. (1951), *Physiology of the Newborn Infant*. Oxford: Blackwell.

ENTEROLITH ILEUS

By W. F. WALKER, G. KERR, and J. S. MACDONALD

DEPARTMENTS OF SURGERY AND RADIOLOGY, UNIVERSITY OF ST. ANDREWS, QUEEN'S COLLEGE, DUNDEE, AND ROYAL INFIRMARY, DUNDEE

ALTHOUGH duodenal and jejunal diverticula are not uncommon, there are few recorded cases where intestinal obstruction has resulted from extrusion of an enterolith into the intestine, with subsequent impaction at a lower level. We have been able to trace only 8 cases recorded so far and wish to report 3 more cases.

CASE REPORTS

Case 1.—Male, aged 58 years. Admitted, with sudden severe abdominal pain of three hours' duration. Five years previously the patient had been investigated for melæna and a barium meal examination carried out. A

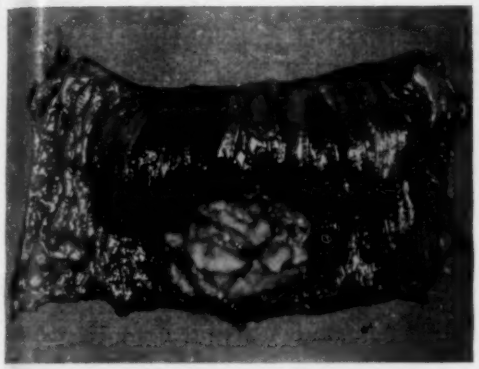

FIG. 172.—Enterolith impacted in lower ileum.

slight irregularity of the duodenal cap had been noted, and the presence in the proximal jejunum of several small fluid levels. In the interval he had been relatively symptom-free. The presence of widespread abdominal tenderness and rigidity on his latest admission led to a diagnosis of perforated duodenal ulcer.

AT OPERATION.—Peritoneal soiling was present owing to a perforation of the ileum at the site of an impacted enterolith, with signs of proximal obstruction (*Fig.* 172). A wedge resection of the perforated bowel was carried out with end-to-end anastomosis.

Post-operative recovery was uneventful. The enterolith was radio-opaque and chemically contained a high percentage of barium. It measured 3 cm. × 2 cm. There can be little doubt that this enterolith was formed in a diverticulum following the barium meal given five years previously.

Case 2.—Male, aged 69 years. Admitted suffering from colicky central abdominal pain and vomiting of two days' duration. He had complained of flatulence, heartburn, and epigastric pain for 18 months. The pain came on 2 hours after meals and was relieved by alkalis. He had also noted occasional abdominal distension and borborygmi for two months prior to admission to hospital. On examination, central abdominal distension was evident and loud bowel-sounds could be heard.

Shortly after admission his symptoms abated and he was treated conservatively for three days, when a recurrence of the symptoms and radiological evidence of intestinal obstruction led to surgical exploration.

AT OPERATION.—The small bowel was distended above a hard mass in the ileum 2 ft. from the ileocæcal valve. The enterolith was removed by enterotomy and was thought to be a gall-stone. The gall-bladder, however, was normal, emptied easily on compression, and did not contain gall-stones. Examination of the rest of the abdomen was negative. The enterolith measured 3·5 cm. × 2·5 cm. and was made up of organic material with small amounts of calcium, much fat, no phosphates, and no cholesterol.

FIG. 173.—Barium loculated in many jejunal diverticula.

The patient reported back four years later complaining of flatulence and abdominal pain. Borborygmi were clearly audible. A barium meal and follow-through revealed the presence of jejunal diverticula (*Fig.* 173). In view of the patient's general condition and age he was treated conservatively.

Case 3.—Female, aged 64 years. On admission this patient complained of sudden onset of severe pain starting in the left loin, radiating down into the left groin and across the hypogastrium. She had had attacks of severe colicky pain in the abdomen for the past 3 to 4 years. During these attacks she often vomited dark coffee-ground material. The gall-bladder had been removed five years previously. There was general abdominal tenderness, most marked in the left iliac fossa. A perforated diverticulitis of the colon was diagnosed and, because of her poor condition, she was treated with antibiotics. She improved at first, but later her condition deteriorated and she died 16 days after admission.

AUTOPSY REPORT (Dr. W. Guthrie).—On opening the abdomen gas escaped together with foul-smelling, orange-green, semi-fluid material. There were many adhesions around the abdominal scar and the omentum was largely located in this region. Several pockets of pus, discoloured by contamination with intestinal contents, were isolated on further dissection. These pockets were related to the small perforations of the jejunum, four in number. Diverticula on the mesenteric border showed varying degrees of inflammation—some, including the largest (3 cm.), contained inspissated debris; a 2·5-cm.

mass of such material was impacted about a metre from the duodenojejunal junction. The gut proximally was dilated, thin, and friable, while distally it was collapsed. The common bile-duct was a thickened (1 mm.) tube, diameter up to 1·5 cm., and contained two gall-stones.

DISCUSSION

Some details of the reported cases of enterolith ileus in association with duodenojejunal diverticula are recorded in *Table I*. The small number of cases that he had seen 8–10 cases with intestinal enteroliths in his practice in 20 years. It is possible that these stones were related to the presence of diverticula, as suggested by our own experience in *Case* 2. It would therefore seem advisable to carry out a barium meal and follow-through in all cases where an enterolith has been found in association with a normal gall-bladder.

When an enterolith has formed in the diverticulum, it normally remains there and is rarely

Table I.—Reported Cases of Enterolith Ileus

Author	Year	Sex	Age (Years)	Site of Obstruction	Operation	Size of Enterolith
Christ	1932	M.	60	Ileum	Enterotomy	6 × 4 × 3 cm.
Hellström	1936	F.	60	Jejunum	Enterotomy	35 × 20 × 18 mm.
Shaw	1940	F.	56	Ileum	Enterotomy	4·5 × 3 cm.
Goodall	1948	M.	57	Ileum	Enterotomy	5 × 2·5 cm.
Armitage and others	1950	F.	73	Ileum	Enterotomy	41 × 21 × 35 mm.
Slater	1953	F.	61	Jejunum	Enterotomy	5 × 3 cm.
Burkitt	1957	F.	56	Ileum	Enterotomy	6·5 × 4 cm.
Blair	1959	F.	76	Jejunum	Resection	4·2 × 4·2 cm.

is rather surprising, in view of the incidence of 4 cases of small-intestine diverticulosis in 300 autopsies reported by Rosedale and Lawrence (1936).

A possible reason for this lies in the factors responsible for the formation of small-bowel enteroliths. One of these factors, stasis, is certainly found in association with diverticula; another, that of chemical composition of the intestinal contents, is variable. Raper (1921) noted that enteroliths commonly contain choleic acid, and Armitage, Fowweather, and Johnstone (1950), discussing the factors responsible for the formation of choleic acid enteroliths, thought that they were unlikely to be produced unless the choleic acid content of the bile was greater than that normally found. They also remark on the greater and almost exclusive prevalence of such stones in women. There are thus two main groups of factors postulated in the formation of small-bowel enteroliths—mechanical and chemical. Added to these is the predisposing factor in some cases of foreign bodies, e.g., fruit skins. *Case* 1 is a good example of a predisposing factor, i.e., barium in a diverticulum representing a late and rather unusual complication of barium meal investigation. Another and perhaps more pertinent reason for this low incidence is that the presence of duodenojejunal diverticulosis is often unsuspected, as it was in *Case* 2. Edwards (1939) found that, even at autopsy, the diverticula could only be demonstrated on occasion by raising the intraluminal pressure or by opening the gut. Knowing this, Mayo, Baskin, and Hagedorn (1952) have advocated the injection of air into the jejunum at operation to distend the diverticula.

Phillips (1921) described 2 cases of intestinal obstructions due to enteroliths. Both patients were women, and in each the pre-operative diagnosis was that of gall-stone obstruction. At operation in one case and autopsy in another, the gall-bladder was found to be normal. The exact cause of the formation of the stones was therefore unknown. He noted extruded, even though the diverticulum usually has a wide neck. While there, it may cause obstruction by its very size—enlarging and encroaching on the intestinal lumen (Terry and Mugler, 1921)—or diverticulitis and perforation of the diverticulum may occur (Slater, 1953). That extrusion of an enterolith into the intestine is unusual must be due to the absence of a strong muscle coat—the diverticulum consisting mainly of protruding mucosa and submucosa with a very thin muscle layer, if any, on the outside. Once an enterolith has been extruded, it behaves in the same way as does a gall-stone, impacting usually in the lower ileum. Enterolith obstruction must therefore enter into the differential diagnosis of gall-stone ileus. Operation with exclusion of gall-bladder disease, and the demonstration of duodenojejunal diverticula at operation or by barium-meal studies afterwards, are the only final means of settling the diagnosis, and confirming the aetiology of the enterolith formation.

SUMMARY

1. Three cases are described where obstruction of the small bowel was due to extrusion of an enterolith from a jejunal diverticulum.

2. The association of small-bowel enteroliths with a normal gall-bladder should suggest the presence of duodenal or jejunal diverticula. If this is not confirmed at operation a barium meal and follow-through should be carried out post-operatively.

3. The incidence of small-bowel obstruction due to an enterolith extruded from a diverticulum may be greater than the literature suggests.

We wish to thank Professor Douglas and Mr. G. M. Sturrock, under whose care the patients were admitted, for advice and for permission to publish these cases. We are also indebted to Dr. W. Guthrie for the pathological report in *Case* 3.

REFERENCES

ARMI AGE, G., FOWWEATHER, F. S., and JOHNSTONE, A. S. (1 50), *Brit. J. Surg.*, **38**, 21.
BLAI J. S. G. (1959), *Lancet*, **2**, 87.
BURK T, R. (1957), *Surgery*, **41**, 867.
CHRIS , A. (1932), *Dtsch. Z. Chir.*, **236**, 561.
EDWA DS, H. (1939), *Diverticula and Diverticulitis of the Intestine*. Bristol: John Wright.
GOOD LL, A. L. (1948), *Brit. med. J.*, **2**, 206.
HELLS RÖM, J. (1936), *Hygiea, Stockh.*, **98**, 480.
MAYO, C. W., BASKIN, R. H. J., and HAGEDORN, A. B. (1952), *Ann. Surg.*, **136**, 691.
PHILLIPS, J. (1921), *Brit. J. Surg.*, **8**, 378.
RAPER, H. S. (1921), *Biochem. J.*, **15**, 49.
ROSEDALE, R. S., and LAWRENCE, H. R. (1936), *Amer. J. Surg.*, **34**, 369.
SHAW, J. J. M. (1940), *Brit. J. Surg.*, **28**, 328.
SLATER, N. S. (1953), *Ibid.*, **41**, 60.
TERRY, W. I., and MUGLER, F. R. (1921), *Arch. Surg., Chicago*, **2**, 347.

GLOMUS TUMOUR OF THE STOMACH*

BY W. S. A. ALLAN AND R. W. S. MILLER

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GLOMUS tumours are well-recognized and characteristic benign neoplasms arising from the neuromyoarterial glomus described by Masson (1924). As such they usually occur in the skin of the extremities, where this structure is most commonly found.

FIG. 174.—Glomus tumour of stomach. Naked-eye appearance.

Rare instances of their occurrence in various other situations have been reported from time to time, and within recent years a small number of tumours has been described in the stomach. The first of these was recognized by de Busscher (1948 a), and to date 15 cases have been recorded, principally from American sources, but also in the Russian and French literature. The report by Kay and his associates (Kay, Callahan, Murray, Randall, and Stout, 1951), who have hitherto been credited with the original description of glomus tumours in this situation, gives an excellent account of the pathological features. The case here described is believed to be the first in the British literature. References to other case reports are given below.

With the exception of 3 cystic tumours, all the examples reported were situated intramurally in the gastric antrum. Some were seen radiologically and half the cases presented with gastro-intestinal bleeding due to ulceration over the tumour. Most specimens measured between 1 and 5 cm. in diameter. Although they were not usually encapsulated, a few were readily shelled out either at operation or on examination of the resected specimen. The sex incidence was equal, the age incidence between 41 and 74 years.

* Received for publication, November, 1959.

CASE REPORT

CLINICAL REPORT.—The patient, a woman aged 72 years, was first admitted to another hospital in 1957 suffering from generalized postprandial abdominal pain, which was accompanied by severe melæna. Clinical examination

FIG. 175.—Compact structure with angiomatous area. H. and E. (× 9.)

elicited only tenderness in the epigastrium and left iliac fossa. Barium enema showed a spastic colon with early diverticulitis. The patient made an uneventful recovery, but shortly after discharge was re-admitted with severe hæmatemesis and melæna. A barium meal showed spasm of the lower end of the œsophagus with possible varices, but the stomach and duodenum were normal. She again responded to conservative treatment and was discharged, no diagnosis being recorded.

Subsequently she was admitted to Bangour General Hospital on March 3, 1959, with a history of severe abdominal pain which preceded a moderately severe hæmatemesis and melæna. There had been no epigastric discomfort or dyspepsia in the few days before the attack.

On examination it was found that bleeding was continuing and her blood-pressure was 70/40 mm. Hg. Treatment was by sedation and the transfusion of 6 pints of whole blood, which raised the hæmoglobin level from 10·5 g. to 11·9 g. per 100 ml.

Three weeks later, on April 1, a further severe hæmorrhage occurred, causing severe melæna and even the passage of red blood per rectum, compelling laparotomy. At operation there was no evidence of peptic ulceration, but a small tumour was felt on the lesser curvature of the stomach about 1 in. from the pylorus. It was assumed

that this was the source of the bleeding and a Polya-type partial gastrectomy was carried out.

Immediate post-operative progress was satisfactory, but on the third day there was a further small hæmatemesis and melæna. Over the next few days altered blood was continually aspirated through a Ryle's tube. The hæmoglobin level was maintained at 11·0 g. per cent by the transfusion of a further 9 pints of whole blood. The bleeding and clotting times were normal; œsophagoscopy revealed no lesion to the level of the cardia.

In view of the continued bleeding, further operation became necessary. On the tenth post-operative day the stomach remnant was opened through a thoraco-abdominal approach. There was a submucous hæmatoma at the distal end of the lesser curvature, but no other lesion was found to account for the bleeding. A more radical gastrectomy was performed to remove a possible source of hæmorrhage from the suture line.

Subsequently her general condition gradually deteriorated. Gastro-intestinal bleeding continued, and despite the transfusion of a further 9 pints of whole blood she died, 15 days after her second operation, on April 25.

NECROPSY REPORT.—Necropsy was performed 6 hours after death. The significant findings were confined to the gastro-intestinal tract.

The mouth, tongue, pharynx, and œsophagus were normal; no varices were found. The gastric stump was empty, but the lower parts of the small intestine and the cæcum were distended with dark clotted blood. The distal half of the colon and the rectum contained scybalous dark fæces.

Inspection of the mucous membrane of the small gastric stump revealed an ovoid ulcer 1·0 cm. × 0·8 cm. on the posterior aspect of the gastro-œsophageal junction. Its floor was covered with necrotic tissue and blood-clot. Microscopically this lesion did not conform to the appearances of a peptic ulcer and appeared to be traumatic; it penetrated only to the submucous layer, with an associated small abscess in the outer part of the gastric wall. It is possible that it was caused by pressure of the Ryle's tube or was due to an isolated erosion of the type found in the Mallory-Weiss syndrome. A second tiny mucosal

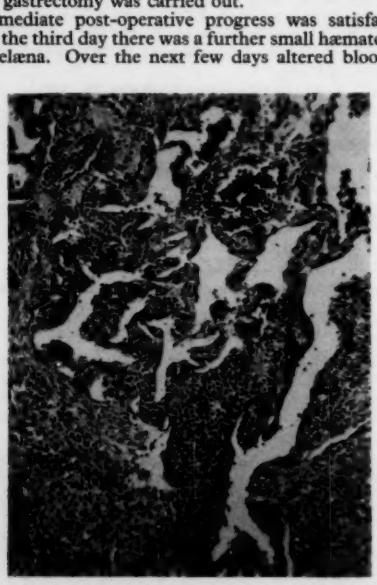

FIG. 176.—Sinusoidal spaces cuffed by glomus cells. H. and E. (× 90.)

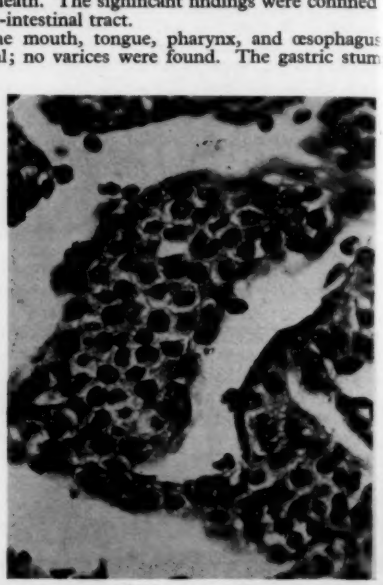

FIG. 177.—High-power view of glomus cells. H. and E. (× 450.)

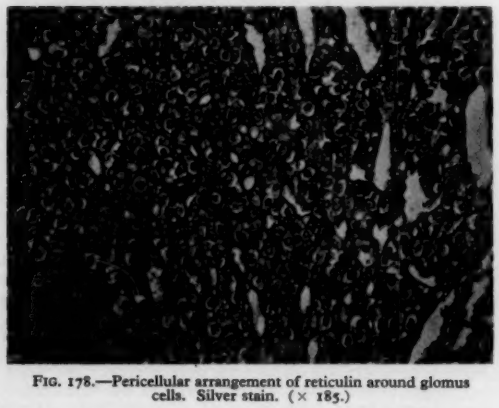

FIG. 178.—Pericellular arrangement of reticulin around glomus cells. Silver stain. (× 185.)

FIG. 179.—Glomus tumour in the submucosa; note the compact structure, lobulation, incomplete encapsulation, and infiltration of muscularis. H. and E. (× 6·7.)

erosion 2 mm. in diameter was found in the fundus of the stomach. Microscopically it also penetrated only to the submucosa and showed a mild inflammatory reaction at its base. The gastric stoma appeared to be normal.

Close inspection of the small intestine revealed no mucosal lesion; the pelvic colon showed a moderate degree of diverticulosis; no hæmorrhoids were demonstrated in the rectum.

It was evident from the necropsy and examination of the second operative specimen that bleeding was due to acute gastric erosions. What was thought to be the source of bleeding at the first gastrectomy proved to be a glomus tumour with no overlying ulceration of the mucosa.

DESCRIPTION OF THE GLOMUS TUMOUR.—

Macroscopic Examination (*Fig.* 174).—The specimen received from the first operation consisted of the distal third of the stomach, including the pylorus. In the antrum 3 cm. from the pylorus there was an intramural swelling on the lesser curvature. The tumour protruded into the gastric cavity, but the mucous membrane covering it was

FIG. 180.—Angiomatous area in submucosa involving the muscularis mucosæ (top left). H. and E. (× 56.)

not adherent or ulcerated. There was no lesion of the mucosa to account for the bleeding.

Section through the tumour disclosed a round, white, firm mass measuring 1·5 cm. in diameter. It appeared to occupy the muscular and submucous layers, but the mucosa was freely movable over it.

Microscopic Examination.—The tumour was well circumscribed and situated in the submucosa. Although it was predominantly solid, one angiomatous area gave a valuable clue to the glomus nature of the condition (*Fig.* 175).

This area consisted of sinusoidal blood-spaces lined by normal endothelial cells. The spaces were separated from each other by several layers of regular rounded cells with centrally placed nuclei. In many of them the cytoplasm was clear, in others it was eosinophilic and finely granular. The regularity of the cells was striking and no mitotic figures or nuclear pleomorphism were seen (*Figs.* 176, 177).

The remainder of the tumour was composed of similar glomus ('epithelioid') cells arranged in compact masses supported by a small amount of fine stroma. Many compressed capillary and sinusoidal spaces lined by normal endothelium lay between the cells. Their relationship to the 'epithelioid' cells was clearly shown in preparations stained for reticulin (*Fig.* 178). This illustration shows that the tumour cells are situated outside the endothelium and do not arise from it. Silver stains also emphasize the vascular nature of the tumour, a point which is less readily appreciated in routine preparations of this rather solid growth. Further, the well-developed pericellular reticulin network illustrates one of the characteristic features of a glomus tumour. No whorled pattern or nerve tissues were present in any of the sections and there was no transition of glomus cells to spindle forms.

A fibrous capsule partly surrounded the growth, merging with condensed submucous tissues; septa arising from its deep surface divided the tumour into lobules (*Fig.* 179). The capsule was infiltrated by tumour, the vessels of which communicated directly with a prominent capillary network in the submucosa (*Fig.* 180). This imparted an angiomatous character to the area, but glomus cells were not present in the vessel walls. The muscularis mucosæ was fragmented and in places entirely replaced by the angioma, but the mucous membrane was unaltered.

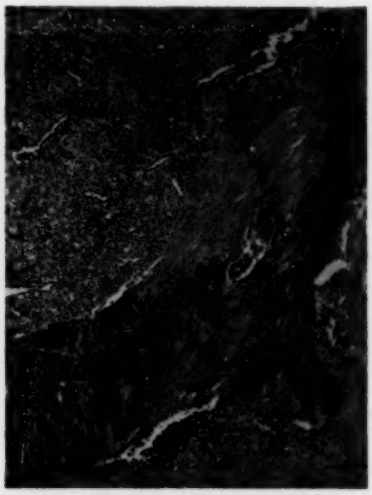

FIG. 181.—Intramuscular infiltration. H. and E. (× 56.)

The deeper part of the tumour was not encapsulated and muscle-bundles undergoing replacement fibrosis were incorporated in its substance (*Fig.* 181).

DISCUSSION

This example of a glomus tumour of the stomach occurred as an incidental finding in a patient presenting with severe hæmorrhage from acute gastric erosions. The mucosa over the growth was intact.

The tumour had a close structural resemblance to those described by Kay and his associates, being composed of both angiomatous and solid tissue. Indeed, all glomus tumours illustrated in the literature have possessed such angiomatous features. The sinusoidal spaces cuffed by glomus cells provide a readily recognizable picture, but such areas may be small and widely spaced, as in this case, the growth being compact and of the paucivascular type described by Dupont (1935) in connexion with cutaneous glomangiomata.

The pericellular arrangement of reticulin around the 'epithelioid' cells is another characteristic feature, and taken in conjunction with the organoid structure helps to differentiate the glomangioma from the related hæmangio-pericytoma.

There are no characteristic features which enable these tumours to be diagnosed clinically or at operation, but once the possibility of their existence in the

stomach is realized, their histological recognition presents no very difficult problems. Those with areas of fusiform cells and a whorled pattern may have to be distinguished from neurogenic neoplasms. A few recorded cases were initially diagnosed on quick-frozen section as carcinoid tumours, but in the routine sections their true nature was readily recognized.

Five of the published cases are sufficiently distinctive to merit comment.

The tumour described by Spängler (1953) was most unusual, consisting of a very large thick-walled cyst containing 1500 c.c. of yellow fluid. Unfortunately a histological description is not given and there is only one photomicrograph, which shows a structure vaguely reminiscent of glomangioma.

In the Russian literature Kostina (1952) described 2 cases, one patient having a cystic tumour 10 cm. in diameter protruding from the greater curvature between the layers of the omentum. It was filled with blood, its wall measured up to 1 cm. in thickness, and from the detailed histological account is readily acceptable as a glomangioma. The other patient had a solid tumour similar to other examples cited in the literature.

Demin (1955) also recorded a thick-walled cystic tumour of similar dimensions to that of Kostina, but gave no histological details.

Of the tumours described by French authors (Lamy and Bonneau, 1956; Rubens-Duval, Baudouin, and Thierrée, 1956), that of Lamy and Bonneau contained a central cystic area with a gelatinous content. Both reports mention a whorled pattern in parts of the tumour, and the occurrence of fusiform cells. These were described as Schwannian elements with nuclear palisading by Lamy and Bonneau. A transition from epithelioid to fusiform cells was noted by Rubens-Duval and his associates. These reports illustrate a neural variant occurring in gastric glomangiomata. Neural elements are recognized in subcutaneous glomus tumours and may constitute the predominant histological feature (Dupont, 1931).

It is difficult to reconcile such patterns with the origin of these tumours from pericytes, discussed below, and with the relation of the glomus cells to smooth-muscle cells. The possibility must be borne in mind, however, of separate proliferation by the nerve elements in the arteriovenous anastomoses from which such tumours are believed to arise.

Histogenesis.—The normal cutaneous glomus is one of the best-known examples of an arteriovenous anastomosis, but similar shunts have been described in many other situations (Clarke, 1938; Clara, 1939). Two structural types have been described (Barlow, Bentley, and Walder, 1951): the direct type in which the connecting channel is thin-walled and controlled by muscular sphincters at each end; and the glomus type, in which a convoluted endothelial-lined vessel is surrounded by a cuff of glomus cells. These cells gradually merge with the muscle cells of the arterial and venous parts of the anastomosis and are believed to control the patency of the shunt.

Examples of the glomus type of anastomosis have been described by Lowenstein (1951) in the choroid coat of the eye, and by de Busscher (1948 b) in the subserous and submucous layers of the stomach. Their distribution was stated to be chiefly in the lesser curvature and posterior wall near the cardia.

Murray and Stout (1942) have put forward strong evidence for the identification of glomus ('epithelioid') cells as Zimmerman's capillary pericytes. The latter are believed to be modified smooth-muscle cells closely applied to the outside of the capillary endothelium. They were demonstrated by Zimmerman in many parts of the body, and the normal neuromyoarterial glomus of the skin derives its characteristic appearance from crowding of the glomus cells around the endothelial channels.

The occurrence of glomangiomata in odd situations has been tentatively explained by the wide distribution of capillary pericytes. In our view these tumours may arise at any site where arteriovenous anastomoses of the glomus type are to be found. This brings the gastric glomangioma more closely into line with its subcutaneous counterpart.

SUMMARY

1. A case of glomus tumour of the stomach is described. This is the sixteenth published case, and the first in the British literature.

2. The importance of opening the stomach where a palpable tumour is found at operation for gastrointestinal bleeding is well illustrated.

3. Glomus tumours are benign neoplasms believed to arise from capillary pericytes present in arteriovenous shunts in the stomach.

We wish to thank Mr. N. A. Gray for permission to publish this case, Professor G. L. Montgomery for his help, and Mr. T. C. Dodds for the photomicrographs.

REFERENCES

* ALLEN, R. A., and DAHLIN, D. C. (1954), *Proc. Mayo Clin.*, **29**, 429.
BARLOW, T. E., BENTLEY, F. H., and WALDER, D. N. (1951), *Surg. Gynec. Obstet.*, **93**, 657.
* DE BUSSCHER, G. (1948 a), *Acta neerl. morph.*, **6**, 87.
— — (1948 b), *Acta gastro-ent. belg.*, **11**, 333.
CLARA, M. (1939), *Die arterio-venösen Anastomosen.* Leipzig: J. Barth.
CLARKE, E. R. (1938), *Physiol. Rev.*, **18**, 229.
* DEMIN, W. N. (1955), *Vop. Onkol.*, **1**, 113.
* DONOVAN, R. J., GRAHAM, J. H., and O'DONNELL, A. R. (1958), *J. int. Coll. Surg.*, **29**, 699.
DUPONT, A. (1931), *Rev. belge Sci. méd.*, **3**, 624.
— — (1935), *Bull. Soc. franç. Derm. Syph.*, **42**, 1260.
* KAY, S., CALLAHAN, W. P., jun., MURRAY, M. R., RANDALL, H. T., and STOUT, A. P. (1951), *Cancer*, **4**, 726.
* KOSTINA, I. (1952), *Trudy Akad. Med. Nauk., Moskva*, **21**, 16.
* LAMY, J., and BONNEAU, H. (1956), *Mém. Acad. Chir.*, **82**, 147.
LOWENSTEIN, A. (1951), *Amer. J. Ophthal.*, **32**, 1651.
* MANNIX, A. J., jun., SCHRAFT, W. C., REED, W. P., and ADIE, G. C. (1955), *Surgery*, **37**, 473.
MASSON, P. (1924), *Lyon. chir.*, **21**, 257.
MURRAY, M. R., and STOUT, A. P. (1942), *Amer. J. Path.*, **18**, 183.
* RUBENS-DUVAL, A., BAUDOUIN, Y., and THIERRÉE, R. (1956), *Mém. Acad. Chir.*, **82**, 152.
* SHOCKETT, E., MOELLER, H. C., CHEATLE, E. L., and TELOH, H. A. (1957), *Gastroenterology*, **32**, 115.
* SPÄNGLER, H. (1953), *Chirurg.*, **24**, 181.

* Case reports of gastric glomus tumours.

SUBMUCOSAL ILEAL GRANULOMA WITH EOSINOPHILIC INFILTRATION AND INTUSSUSCEPTION

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THIS is the report of a submucosal granuloma of the ileum with eosinophilic infiltration resulting in intussusception with subacute obstruction. Such lesions of the gastro-intestinal tract are rare. In a recent overall review of 49 cases by Toole and Moscopoulos (1959), only 9 have occurred in the small intestine, of which 2 were located in the ileum. The following patient is an addition to these latter cases.

CASE REPORT

A 74-year-old white woman complained that she had been unwell for about six weeks. There were no definite symptoms, but she did not feel herself. She had lost weight and her daughter said that at times she passed dark, but not black, fæces. Ten days prior to hospitalization, vague, colicky abdominal pain with intermittent vomiting developed, and then admission to hospital became necessary.

ON ADMISSION.—The pain was located low in the right side of her abdomen and vomiting occurred twice a day. Her bowels were open daily. Examination revealed a pale, dehydrated, elderly woman with minimal distension of the abdomen, no visible peristalsis, but tenderness and guarding to palpation in the right iliac fossa. No mass was palpable. Rectal examination revealed no abnormality. A vaginal examination was not performed.

SPECIAL INVESTIGATIONS.—Plain radiograph of the abdomen: Some dilated loops of small intestine with fluid levels.

Blood-picture: Hb 50 per cent, R.B.C. 2,200,000, W.B.C. 8300. Differential: Neutrophils 66 per cent, eosinophils nil, lymphocytes 32 per cent, monocytes 2 per cent.

Blood-urea: 71 mg./100 ml.

Occult blood: Positive.

Temperature: 98°–99° daily.

The patient was initially put on a drip and suction régime and her anæmia improved after 2 pints of blood. Following these measures, the vomiting ceased and her abdomen became less distended. She had now been in hospital one week and examination of the abdomen revealed in the right iliac fossa a mass 3 in. × 2 in., mobile, non-tender, and approximately 2 in. from the anterior superior iliac spine in the horizontal plane. The occult blood-tests were persistently positive and her anæmia could only be controlled by transfusion. Laparotomy was decided upon.

OPERATIVE FINDINGS.—Exploration revealed in the proximal ileum a mass which was stuck below to the bladder and to the distal ileum. When the adhesions were separated it was found that the proximal ileum had intussuscepted into the distal ileum for about 3 in. There were no nodes palpable in the mesentery. Resection of the intussusceptum and intussuscipiens was performed with restoration of continuity by end-to-end anastomosis.

One week post-operatively a bladder fistula developed, but with an in-dwelling catheter this closed spontaneously. A further complication was an inferior myocardial infarct, from which a satisfactory recovery was made without the use of anticoagulants.

PATHOLOGICAL REPORT ON SPECIMEN.—

Macroscopically.—A pedunculated tumour, watery yellow in colour, forming the apex of an intussusception with upper ileum invaginated into lower.

Microscopically.—The ileal tumour appears to be homogeneous and is a submucosal granuloma. It is composed of uniformly distributed fibroblasts in an œdematous stroma infiltrated with eosinophil cells of characteristic morphology. There are some small round cells and plasma cells but few neutrophils. Fat staining and polarography

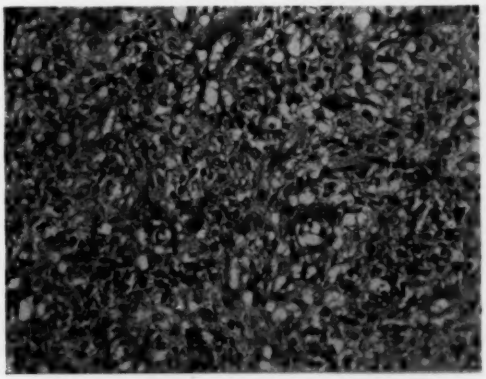

FIG. 182.—The spaces are œdema spaces only. Staining revealed no lipoid. The cells with dark nuclei in this picture are mainly eosinophils.

failed to reveal any lipoid. The condition differs from the eosinophilic xanthomatous granuloma commonly found in bones in that there are no histiocytes, no giant cells or macrophage storage, no xanthomatous reaction, and fibrosis is minimal (Thannhauser, 1958). (*Fig.* 182.)

Following this report further investigations were undertaken to distinguish the lesion from true eosinophilic granuloma.

1. Radiography of the skeletal system revealed no bony abnormality.

2. Blood-films: No eosinophilia in the immediate post-operative period, but five months later had risen to 17 per cent.

3. Biopsy of nodes in the axillæ revealed lipomelanosis only.

4. Liver and spleen were not palpable.

5. Chemical examination of serum constituents:
Total cholesterol level: 90 mg. per cent. Low.
Fatty acid level: 6·0 mEq./l. Normal.
Beta-lipoprotein: 8·0 flocculation units. Normal. No pre-beta fraction.
Hæmoglobin: 13·4 g. per 100 ml.
Accelerated clotting time: 16 sec. Normal.
E.S.R.: 43 mm. per hr.
Kunkel flocculation test for gamma globulin: 13 units. Slightly raised.

6. Skin sensitivity tests: Positive for fish, shell-fish, and cereals. These were not considered specific.

Her recovery has been complete and the patient is living a normal life for her age.

COMMENTS

When the patient responded to transfusion and the mass in the right iliac fossa became palpable it was considered that this was a carcinoma of the cæcum.

In view of her frailty neither a barium meal and follow-through nor an enema was considered advisable as there was the risk of increasing the obstruction and jeopardizing her chance of survival. At operation the granuloma resembled macroscopically a pedunculated lipoma which had initiated the intussusception. It was only on section that the nature of the tumour was suspected. Then remained the necessity of differentiating the tumour from a true eosinophilic granuloma which belongs to the group of disorders classified under the heading of 'medullary reticulosis' (Robb Smith, 1938). This has been done conclusively by section (*see above*) using special fat-staining technique and polarography; with the latter there was no double refraction characteristic of cholesterol esters and staining was negative for lipoid.

A granuloma in the ileum is rare. The lesion occurs most commonly in the prepyloric region of the stomach and may or may not be associated with lesions of the intestines. The first extensive description was by Herrera and De La Guardia in 1948. They described a lesion of the pylorus, yellow-green in colour, soft and friable, and it extended along the whole length of the small bowel as annular thickenings. There was no mesenteric lymphadenopathy, but a post-operative eosinophilia of 20 per cent developed on the seventh day. In the reported cases such widespread involvement is not a common occurrence. The lesions tend to be confined to the pylorus and duodenum and rarely extend to or involve separately the jejunum, as in the case of Polayes and Krieger (1950). This is the first case described of localized eosinophilic granuloma of the intestine.

The microscopical picture has been best described by Vaneck (1949), who stated that there were three essential components of the lesion:—

1. That fusiform fibroblastic cells are present with a loose network of collagenous fibres and through which lymphocytes are distributed. These may be grouped into rudimentary lymph-follicles.

2. That eosinophilic polymorphonuclears are distributed throughout the tissues, and most marked in the deep submucosa and muscularis. These may break up the muscle-bundle into bands, with the appearance of cavities of various sizes from microscopic to gross proportions.

3. That the cavities be filled with eosinophilic œdematous material. He thought that it was probably an inflammatory lesion of chronic granulomatous character rather than a fibroma with secondary inflammatory reaction.

Although both diffuse and localized lesions have been described, Booher and Grant (1951) state that the commonest manifestation of the disease is the formation of a poorly circumscribed submucosal nodule which tends to become pedunculated and polypoid, and that it occurs most frequently in the pyloric antrum. However, the pyloric sphincter does not seem to be the limit of the extension in a few cases.

Whether this is an allergic manifestation or not is still open to question. In 1948 Barrie and Anderson described the case of a 27-year-old woman with concentric hypertrophy of the muscular coat of stomach, pylorus, and duodenum with massive eosinophilic infiltration of the pylorus, associated with peculiar pararterial giant-cell follicles. A true organic, intestinal, allergic reaction was suspected as she was intolerant to certain foods and had a constant blood eosinophilia associated with localized tissue eosinophilia of the pylorus. Eosinophilic granuloma of bone, skin, and lungs differs pathologically from the case reported and differentiated as stated above. The final suggestion by Toole and Moschopoulos (1959) is whether it is in fact due to some metabolic disorder similar to Hand-Schüller-Christian disease and the Letterer-Siwe syndrome.

SUMMARY

This is the report of a submucosal ileal granuloma with eosinophilic infiltration in an elderly woman. It is found more commonly in the pyloric region of the stomach and duodenum and less commonly in the large and small intestine. A pre-operative diagnosis can only be suspected and proved by the subsequent histology. There may, or may not, be an associated allergic state.

I can find no report of the lesion being or becoming malignant, and resection with restoration of intestinal continuity is the treatment of choice.

My grateful thanks are extended to Mr. R. Parkinson for allowing me to publish this paper and for his help. Also to Mr. A. J. Heriot for his valued advice, to Dr. J. B. Enticknap for the detailed histology, and to all those who have helped make this publication possible.

REFERENCES

BARRIE, H. J., and ANDERSON, J. C. (1948), *Lancet*, **2**, 1007.
BOOHER, R. J., and GRANT, R. M. (1951), *Surgery*, **30**, 388.
HERRERA, J. M., and DE LA GUARDIA, J. (1948), *Arch. Hosp. Santo Tomas*, **3**, 19.
POLAYES, S. H., and KRIEGER, J. L. (1950), *J. Amer. med. Ass.*, **143**, 549.
SMITH, A. H. T. ROBB (1938), *J. Path. Bact.*, **47**, 457.
THANNHAUSER, S. J. (1958), *Lipidoses: Diseases of Intracellular Lipid Metabolism*, 3rd ed. New York and London: Grune & Stratton.
TOOLE, H. J., and MOSCHOPOULOS, A. N. (1959), *Brit. J. Surg.*, **46**, 445.
VANECK, J. (1949), *Amer. J. Path.*, **25**, 397.

PAPILLARY ADENOCARCINOMA OF THE FEMALE URETHRA

A CASE REPORT

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"PRIMARY carcinoma of the peri-urethral tissues in the female is a rare disease, and little has been written about it" (Battle, 1895). Six years later (1901) he reported another case, and the histology of the growth was described by Shattock as a "papilliferous columnar-celled carcinoma, the epithelium lining its recesses and processes being of a typical cylindrical form without overlying flat cells". The latter also described, in one of Boyd's (1906) two cases, another tumour with similar histological features. The present case further illustrates the characters of these tumours.

FIG. 183.—Cut surface of excised specimen showing the growth (T) situated between the urethra (indicated by arrow) and the anterior vaginal wall. (× ⅔.)

CASE REPORT

HISTORY.—A Chinese female, aged 60 years, complained of whitish vaginal discharge for 1 year and urethral bleeding for 2 months. Discharge was profuse, whitish, and foul. There was no pain on micturition and the flow of urine was normal. Her menopause started 12 years previously.

After admission a small rupture through the anterior vaginal wall was noted and pressure on the anterior vaginal wall caused a small amount of soft, pinkish-red tissue to appear through the ruptured area and also at the urethral meatus. Biopsies of tissues from both sites revealed a well-differentiated papillary adenocarcinoma.

EXCISED SPECIMEN.—This consisted of uterus, vagina, part of vulva, the whole urethra, and a small portion of the adjoining bladder wall. Except for the urethra and its surrounding tissue, the other parts of the specimen showed no noteworthy changes. In the posterior aspect of the urethra, about 1½ cm. below the internal urethral orifice, there was a hole in the mucous membrane about 2 mm. in diameter which communicated with a cystic area 2 cm. in main diameter. This area was located between the urethra and the anterior vaginal wall (*Fig.* 183), and was occupied by soft, friable, pinkish tumour tissue and plentiful mucoid material. The tumour was attached to a large part of the inner surface of this cystic area. At other places the inner surface appeared smooth. Posteriorly the growth had penetrated the adjoining anterior vaginal wall (*Fig.* 184), the defect measuring 3 mm. in diameter. Further examination revealed that the growth had also invaded through another part of the posterior urethral wall 2 mm. behind the urethral meatus, forming a granular area 3 mm. in diameter. The remaining portions of the urethra appeared normal.

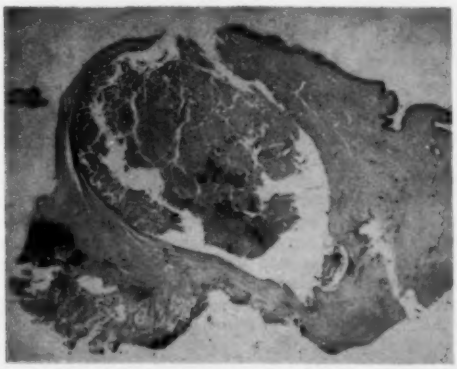

FIG. 184.—A very low power view of the whole growth taken at the site of rupture in the anterior vaginal wall. H. and E. (× 2.)

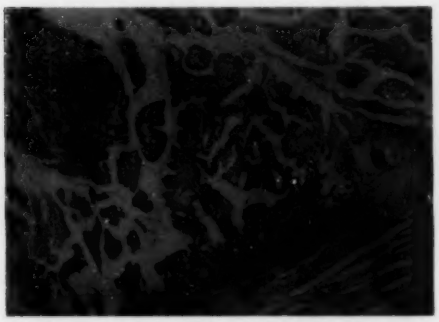

FIG. 185.—A representative field showing features of a well-differentiated papillary adenocarcinoma. H. and E. (× 20.)

Histology.—Three blocks were taken, one of them a cross-section of the whole tumour (*Fig.* 184). The growth lying within the cystic area has the features of a well-differentiated papillary adenocarcinoma (*Fig.* 185). The

papillary processes and glandular acini are lined mainly by tall, columnar, non-ciliated epithelial cells which are closely packed together. Their nuclei vary in size and shape but are mostly moderately large, irregularly ovoid, and not hyperchromatic. Some are vesicular and contain nucleoli. Mitotic figures vary from scanty to moderate in number. The cytoplasm varies from pale pinkish to clear and vacuolated. Mucicarmine stain shows areas where the cytoplasm of these cells contains abundant mucin, sometimes in globules. The core of the papillary processes consists of scanty, loose connective tissue infiltrated by lymphocytes, plasma cells, and few eosinophils.

The wall of the main cystic area in other parts is lined by only a single layer of low or tall, columnar, non-ciliated epithelial cells. These cells are compactly arranged and their cytoplasm contains plentiful mucin. At places intra-epithelial gland formation is seen. There are also areas where the epithelial cells show short papillary infolding or short irregular glandular formation.

Except for extension of the growth through the anterior vaginal wall and the floor of the urethra, there is no invasion of the tumour cells into the adjoining fibromuscular stroma, which is clearly demarcated.

COMMENT

Huffman (1948) made a thorough study of the para-urethral ducts in the adult human female urethra by means of serial sections and wax casts on 11 necropsied specimens. He found that these ducts are not constant in number and location and that they not only form extensive ramifications throughout the tissues about the distal urethra, but also extend to within a short distance of the bladder, the largest of these most distal para-urethral tubules being Skene's duct. Often numerous ducts terminate in tubular glands lined by low or tall columnar cells. Occasional nests of mucus-secreting cells are demonstrated. He also noted that cystic dilatation of the ducts is common. These features are similar to those seen in the present tumour. The origin of the cystic area containing the growth needs further consideration. It may be either a cystic dilatation of the para-urethral tubules as described by Huffman (1948), or a diverticulum. An adenocarcinoma arising from the latter has been described by Hamilton and Leach (1951). Their case received X-ray irradiation prior to removal; but, in spite of this, areas showing spaces resembling acini were seen, mostly lined by cuboidal or columnar epithelium, and scanty mucinous material was demonstrated by mucicarmine stain.

The homologues of the para-urethral glands in males are the glands of Littre; and a tumour arising from these was described by Dobos, Downing and Ashe (1954), but the histological picture of the growth was not an adenocarcinoma.

In a critical review of adenocarcinoma of the female urethra by Walker and Huffman (1947) only 27 unequivocal cases were collected, of average age 53·5 years. They noted that these tumours usually arose from the floor of the urethra, as also in the present case.

The term 'adenocarcinoma of the female urethra' is strictly not correct, because the urethral epithelium gives rise to either epidermoid or transitional-cell carcinoma, whereas the adenocarcinomas arise from the lining epithelium of para-urethral tubules or glands, a distinction comparable to that between epidermal and sweat-gland tumours.

SUMMARY

A well-differentiated papillary adenocarcinoma, located between the urethra and the anterior vaginal wall, and arising from the lining epithelium of either the para-urethral tubule or gland, has been described.

REFERENCES

BATTLE, W. H. (1895), *Lancet*, **1**, 1512.
— — (1901), *Ibid.*, **2**, 79.
BOYD, F. N. (1906), *J. Obstet. Gynæc. Brit. Emp.*, **9**, 40.
DOBOS, E. I., DOWNING, S. W., and ASHE, S. M. P. (1954), *Cancer*, **7**, 539.
HAMILTON, J. D., and LEACH, W. B. (1951), *Arch. Path. (Lab. Med.)*, **51**, 90.
HUFFMAN, J. W. (1948), *Amer. J. Obstet. Gynec.*, **55**, 86.
WALKER, L. M., and HUFFMAN, J. W. (1947), *Quart. Bull. Northw. Univ. med. Sch.*, **21**, 115.

A CASE OF BILATERAL TUBULAR ADENOMA OF THE TESTIS

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AND R. SALM
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TUBULAR adenoma of testis is rare, occurring in about 0·4 per cent of testicular growths (Dixon and Moore, 1952). The following case is presented on account of its rarity and other unusual features.

CASE REPORT

The patient was first seen in April, 1955, at the age of 8 years, for bleeding per rectum. He was backward and enuretic, but able to speak. It was noted that both testicles were considerably enlarged and his parents disclosed that they had been large at birth and had been gradually enlarging over the years. Physical and laboratory examination gave no help in diagnosis; a barium enema was negative, but sigmoidoscopy showed several coiled, grossly distended submucous veins 10 cm. above the anus. No further bleeding having taken place, the patient was discharged, but his parents were advised to report back if it recurred.

He was next seen again in July, 1957, at the age of 10 years, as his scrotum had continued to enlarge (*Fig.* 186). The right testicle was the larger and biopsy revealed a tubular adenoma; a right orchidectomy was performed. Healing of the wound was uneventful, but subsequently a staphylococcal abscess developed in the right groin, necessitating incision and drainage; he was seen at regular intervals. The left, originally the smaller, testis continued to enlarge. In January, 1958, it had attained a size exceeding that of the right testis when it was removed, so a left orchidectomy was performed.

AUXILLARY INVESTIGATIONS.—Nuclear sexing of skin confirmed his male sex. Male toad pregnancy test was negative. The urinary 17-ketosteroids were 7·27 mg. per 24 hr. (normal males 9–24 mg.). The urinary 11-oxycorticosteroids were 1·15 mg. per 24 hr. (normal males 2·6 mg.) (the latter values are approximate only as allowances had to be made for loss due to enuresis). Œstriol was nil (normal in the male). Radiological examination of the skull showed a normal pituitary fossa.

MACROSCOPICAL EXAMINATION OF SPECIMENS.—The right testicle measured 7·3 × 4·8 × 3·9 cm. and weighed 90 g. Normal values at puberty about 3 × 2 × 1·6 cm., 15 g.) It was surrounded by an intact tunica albuginea.

FIG. 186.—Both testicles are considerably enlarged, the right being much larger than the left.

FIG. 187.—Bisected right testicle, showing fleshy, uniform cut surfaces. The discoloured area at the periphery is due to biopsy trauma.

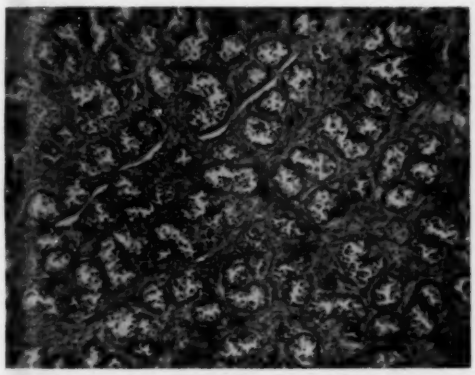


FIG. 188.—Low-power view showing tubules set in a cellular fibrous stroma. H. and E. (× 56.)

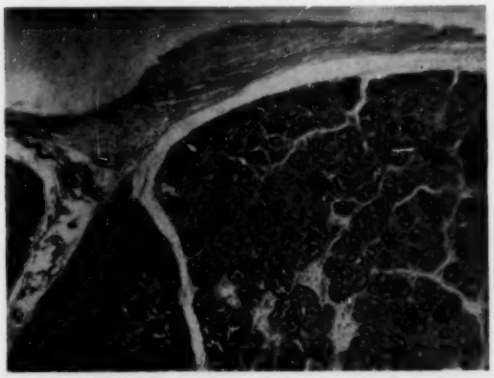


FIG. 189.—Low-power view showing regular tubular pattern, subdivided by fibrous septa and surrounded by tunica albuginea. H. and E. (× 24.)

A tiny epididymal remnant was identifiable, measuring only a few millimetres in length. The cut surfaces were fleshy, uniform, of a small-lobular character of regular pattern, and greyish-pink in colour (*Fig.* 187). No normal testicular tissue was discernible. The left testicle measured 8 × 5 × 4·5 cm. and weighed 100 g. and was otherwise identical in appearance and histology. No fluorescence was noted when the cut surfaces were exposed to ultra-violet light.

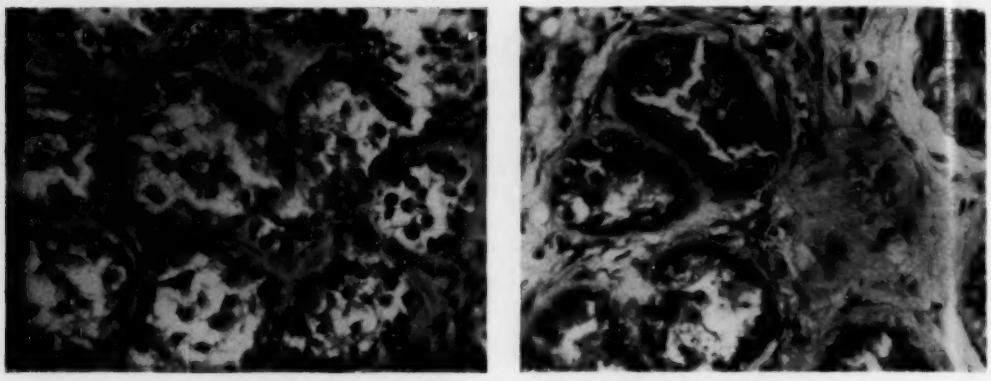
HISTOLOGICAL EXAMINATION.—Several blocks were cut from each specimen. All the sections showed a uniform structure; the tissues consisted of glandular tubules of uniform calibre (*Fig.* 188), set in a matrix of fibrous stroma traversed by thick fibrous septa. The outer surface was covered by an intact tunica albuginea (*Fig.* 189). Here and there were areas of fibrosis associated with tubular degeneration. All the tubules were lined by a single row of cells of Sertoli type with round, basally placed nuclei with evenly distributed chromatin and vacuolated cytoplasm with indistinct margins, which appeared frequently to be drawn out into the tubular lumen (*Fig.* 190 A). No intracellular periodic-acid–Schiff-positive material was demonstrable; frozen sections displayed only a few fine, scattered sudanophil droplets within the epithelial cytoplasm as well as some aggregates of coarser droplets in the stroma. No other cell type was discernible within the tubules. The intertubular connective tissue contained some clumps of poorly defined cells with vesicular nuclei, presumably representing abortive Leydig cells (*Fig.* 190 B). The epididymal tubules themselves were normal, but were lying embedded in congested, œdematous fibrous stroma with some perivascular round-cell infiltration.

DISCUSSION

Tubular adenomata have been recognized since they were first described by Pick (1905). Stalker and Hendry (1952) were able to collect 17 cases from the literature, to which they added 1 case of their own.

These occurred in undescended testes, 6 in otherwise normal males, and 11 in pseudohermaphrodites. In 4 cases the lesions were bilateral. All the patients were postpubertal, their ages ranging from 19 to 54 years. Dixon and Moore (1952), in an American Army series of 990 testicular tumours, found that 5 were

The present case differs in several aspects from those previously recorded. The patient is a very backward, enuretic young boy of male nuclear sex who presented with scrotal testes. As far as we are aware, this is the first time that a tubular adenoma has been recorded before puberty; and moreover the



A B

FIG. 190.—Two high-power fields showing tubules lined by a single row of Sertoli cells. In B, to right of centre, clumps of poorly preserved Leydig cells. H. and E. (× 255.)

testicular adenomata. In 1 case the finding of microscopic tumour in an undescended testis was incidental, but in 4 cases there were associated other testicular growths, namely, a seminoma, an interstitial cell tumour, a choriocarcinoma, and a teratoma; the former two in cryptorchids, the latter two in scrotal testes.

Identical tumours have, in rare instances, been recorded in the female, but as Willis (1959) has pointed out, these cases had been published before the method of nuclear sexing had been introduced. Since, Willis has twice been able to prove in this way that the presumed females were, in fact, pseudohermaphroditic males.

There is general agreement that tubular adenomata represent growths arising from Sertoli cells; Stalker and Hendry (1952) convincingly demonstrated in their case the transition from atrophic seminiferous tubules, lined solely by Sertoli cells, to neoplastic tubules. In man this neoplasm is benign, of infrequent occurrence, and hormonally inactive. One patient, it is stated, presented on account of gynæcomastia which only incompletely regressed after removal of the testicular growth (Teilum, 1946). Neoplasms of this type are not as rare in dogs and may be malignant, causing intra-abdominal metastases. There is evidence of a markedly increased œstrogen production, such as bilateral symmetrical loss of hair, even amounting to complete baldness; enlargement and pigmentation of nipples; laxity of the penile sheath, and perverse attraction for other dogs (Coffin, Munson, and Scully, 1952, and our own observation). Many of these signs may disappear after removal of the diseased, cryptorchid gonad, only to reappear with the formation of hormone-secreting secondaries. Scully and co-workers (1952) demonstrated intracellular steroid hormones, in their case undoubtedly œstrogen, by histochemical methods.

neoplasm was bilateral, being recorded only four times previously, and involved scrotal testes, an occurrence not previously observed unless the growth was combined with other testicular neoplasms (Dixon and Moore, 1952).

The rate of enlargement of the left testicle could be determined with some accuracy. In July, 1957, it had measured (clinically) 7 cm. across; in January, 1958, the excised testis had a transverse diameter of 8 cm.

Tubular adenomata are usually described as small, single, or multiple, benign neoplasms lying in otherwise normal testicular tissue and occurring, as already mentioned above, in cryptorchids. In the present case the entire gonads had been transformed into a bilateral tumefaction of regular pattern, reminiscent of a fœtal testis, with scattered clumps of abortive Leydig cells in the fibrous intertubular stroma, without any trace of normal testicular parenchyma being demonstrable. The minute size of both epididymes is interesting as they corresponded to those of a neonate, although the microscopical structure proved them to be normal.

SUMMARY

A case of bilateral tubular adenomas of scrotal testes is recorded, occurring in a mentally retarded, enuretic boy. After removal of the right testicle, the left was observed to enlarge at a steady rate. On examination the entire testicular masses presented the features of tubular adenoma, no normal testicular tissue being demonstrable. The epididymes were of minute size, though microscopically normal.

Our sincere thanks are due to Professor H. A. Willis for confirming the diagnosis and reading the manuscript. We are most grateful to Mr. J. W. Walker for the biochemical estimations and to Miss Phyllis E. Coleman for help with the photography.

REFERENCES

COFFEY, D. L., MUNSON, T. O., and SCULLY, R. E. (1952), *J. Amer. vet. med. Ass.*, **121**, 352.

DIXON, F. J., and MOORE, R. A. (1952), "Tumors of the Male Sex Organs", pp. 120–126, *Atlas of Tumor Pathology*. Washington, D.C.: Armed Forces Institute of Pathology.

PICK, L. (1905), *Arch. Gynäk.*, **76**, 191.

STALKER, A. L., and HENDRY, W. T. (1952), *J. Path. Bact.*, **64**, 161.

TEILUM, G. (1946), *Acta path. microbiol. scand.*, **23**, 252.

WILLIS, R. A. (1959), in *Modern Trends in Pathology* (ed. D. H. Collins), Ch. 7. London: Butterworth.

A COMPARISON OF SUPRATENTORIAL INTRACRANIAL MENINGIOMAS OPERATED UPON WITH AND WITHOUT INDUCED HYPOTHERMIA

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SINCE the introduction of induced hypothermia as an aid to operative surgery (McQuiston, 1949), the method has been widely and increasingly used. The application of hypothermia to neurosurgery in particular has received a great deal of attention and many claims have been made for the advantages conferred (Botterell, Laughheed, Scott, and Vandewater, 1956).

We present here a series of 120 cases of intracranial, supratentorial meningiomas all admitted under the care of one of the authors (W. McK.) either to the National Hospital, Queen Square, or the Department of Neurosurgery of St. George's Hospital, between 1950 and 1959. Sixty of these patients were operated upon with induced hypothermia and 60 of them under general anæsthesia alone. For both groups of patients the surgical and anæsthetic teams were virtually the same, and the cases were unselected and nearly consecutive. In both groups induced hypotension was routinely used. There is thus afforded an opportunity to make an objective assessment of what differences, if any, the use of hypothermia has made in the management of meningiomas.

The Patients.—These patients presented all the diversity of neurological disturbances which may be caused by meningiomas. As would be expected, they varied from those without mental impairment or neurological signs at one extreme, to the demented and severely disabled at the other. However, nearly all were alert or relatively so, only one patient in the 'normothermic' group being described as 'drowsy' and one in the 'hypothermic' group being in coma (which ensued immediately following ventriculography) at the time of operation. Both of these patients made an excellent recovery. A detailed clinical account of every patient would not be relevant to this article and is therefore not given.

In order to establish that the two groups of patients may be fairly compared, both are presented in tabular and graphic form, so that age, sex, and anatomical site may be clearly seen (*Table I*, *Fig.* 191). These curves demonstrate the well-known

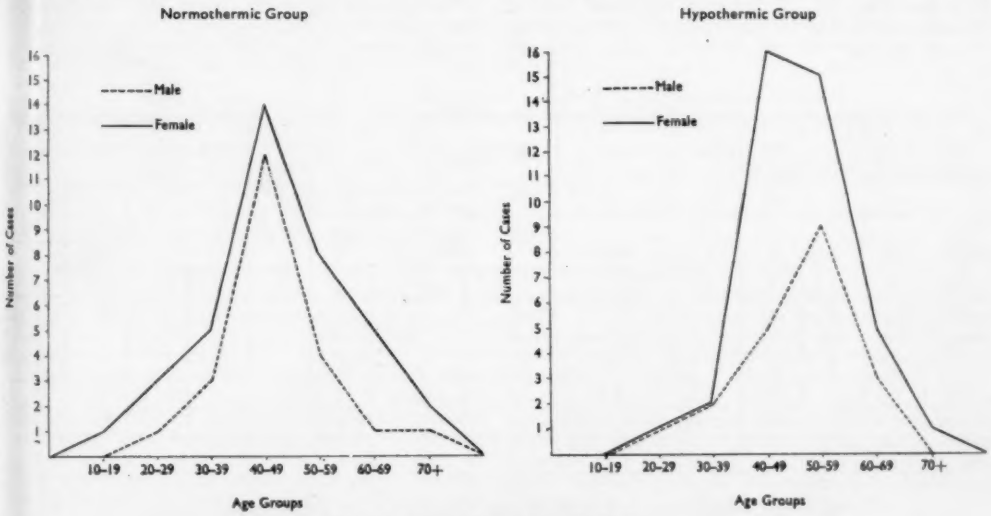

FIG. 191.—Curves of age and sex distribution.

and characteristic age and sex distribution of meningiomas and show but small differences between the two groups.

Table I.—Table of Age and Sex Distribution

Age	Normothermia		Hypothermia	
	Male	*Female*	*Male*	*Female*
10–19	0	1	0	0
20–29	1	3	1	1
30–39	3	5	2	2
40–49	12	14	5	16
50–59	4	8	9	15
60–69	1	5	3	5
70+	1	2	0	1
Totals	22	38	20	40

The anatomical situations of the tumours, correlated with age and sex, are set out in *Tables II, III*. In these tables the incidence of any given type of meningioma may be seen at a glance in either series. It may be pointed out, however, that when the tumours are collectively subdivided into broad 'basal' and 'non-basal' groups the figures are as in *Table IV*. On this basis, therefore, the two groups of patients are seen to be very similar.

Technique of Anæsthesia and Induced Hypothermia.—The methods of induction of hypothermia have been much discussed elsewhere (Hellings, 1958). A brief statement only is made here of the technique used as standard throughout the series. General anæsthesia is induced with thiopentone, pethidine, and a relaxant, and maintained with nitrous oxide, oxygen, pethidine, and trilene as required. Hypothermia is induced by surrounding the patient with ice bags. Drugs such as chlorpromazine, promazine, and promezathine were occasionally used at first and were given up later as an aid to induction of hypothermia. The temperature fall is observed and measured with thermocouples placed in the rectum and œsophagus. A temperature of 30° C. has been aimed at as the lowest desirable level and regularly achieved, an 'after-fall' of up to 2° C. having occurred in a few patients. In both groups of cases controlled hypotension was employed with a systolic blood-pressure of 60–90 mm. Hg as the target. Standard hypotensive agents were used—pentamethonium iodide and hexamethonium bromide initially and until the

Table II.—Anatomical Situation and Tumour Type—Normothermic Group

Age	Parasagittal and Falx		Cortical		Pterional		Olfactory Groove and Subfrontal		Sphenoid Wing		Suprasellar		Subtemporal	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
10–19	0	0	0	0	0	0	0	0	0	0	0	0	0	1
20–29	0	1	1	2	0	0	0	0	0	0	0	0	0	0
30–39	2	2	1	1	0	0	0	0	0	0	0	2	0	0
40–49	6	7	3	3	0	0	0	0	2	1	0	2	1	1
50–59	1	3	1	3	0	0	1	0	1	1	0	1	0	0
60–69	1	2	0	0	0	1	0	1	0	1	0	0	0	0
70+	0	0	1	1	0	1	0	0	0	0	0	0	0	0
Totals	10	15	7	10	0	2	1	1	3	3	0	5	1	2
	25		17		2		2		6		5		3	

Table III.—Anatomical Situation and Tumour Type—Hypothermic Group

Age	Parasagittal and Falx		Cortical		Pterional		Olfactory Groove and Subfrontal		Sphenoid Wing		Suprasellar		Subtemporal	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
10–19	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20–29	0	0	1	0	0	0	0	0	0	1	0	0	0	0
30–39	0	0	0	2	1	0	1	0	0	0	0	0	0	0
40–49	2	3	2	4	1	0	0	2	0	4	0	2	0	1
50–59	3	11	2	3	1	0	2	1	0	0	0	0	0	0
60–69	1	2	0	1	1	0	0	1	1	0	0	1	0	0
70+	0	0	0	0	0	0	0	0	0	1	0	1	0	0
Totals	6	16	5	10	4	0	3	4	1	6	0	4	0	1
	22		15		4		7		7		4		1	

introduction of trimetaphan camphorsulphonate (arfonad) in 1953, since when the latter drug has been used exclusively.

The Operations.—It was hoped at the outset of the series of cases which were cooled for operation, that the reduction of intracranial pressure and brain

Table IV.—SUBDIVISION INTO 'BASAL' AND 'NON-BASAL' TUMOURS

	BASAL	NON-BASAL
Normothermia	16	44
Hypothermia	19	41

volume, reduction of blood-flow, and lowering of blood-pressure and pulse-pressure would result in greatly improved conditions for intracranial surgery. This hope was to some extent realized. Favourable impressions and recollections, however, are not entirely supported by reference to the operation records. It can be said that intracranial pressure and dural tension were described as 'high', 'very high', or 'extremely high' in 16 of the 60 operations done without hypothermia and in 11 of the 60 operations done with induced hypothermia. Figures for hæmorrhage and blood-loss are more impressive, bleeding being described as 'severe' in 13 of the normothermic operations, 11 of the patients requiring blood transfusion; whereas 'severe' hæmorrhage occurred in 5 of the hypothermic operations, only 2 of these patients requiring blood transfusion. The almost immediate fall in blood-pressure, often brought about by relatively small blood-loss in the cooled patient, although slowly compensated, was well tolerated.

In addition to these factors it was considered that diathermy coagulation of blood-vessels was more effective with cooled tissues and that dissection of tumour from the surrounding brain was easier than was the case in normothermic conditions.

Disadvantages of induced hypothermia were few. Occasionally it was found difficult to oxygenate patients adequately, the colour of arterial and venous blood being imperfect and remaining so throughout the procedure, despite all efforts. Such a state of affairs was usually associated with cerebral congestion and raised intracranial pressure. By far the most important drawback, however, was the difficulty experienced in making clinical assessments post-operatively while the patient was still cold. This difficulty usually remained a source of anxiety until normal temperature was approached. Although some patients were fully conscious and co-operative at a temperature of 30°–32° C., this was not the rule, and despite the fact that complications were few it was considered that signs of an early, post-operative clot-compression might well be masked for a significant period of time.

Post-operative Course.—

Mortality.—

A. *Normothermic group*: Four of these 60 patients died as a result of operation:—

1. Male, aged 47 years, with a parietal, cortical meningioma, who died 8 weeks later from infection and intracranial suppuration.

2. Female, aged 52 years, with a parietal, cortical meningioma who died within 48 hours from cerebral infarction.

3. Female, aged 56 years, with a sphenoidal wing meningioma who died within 48 hours from cerebral infarction.

4. Male, aged 44 years, with a parietal parasagittal meningioma who died 3 hours post-operatively from sudden cardiovascular collapse.

Post-mortem examination was made in all cases.

B. *Hypothermic group*: Three of the 60 patients died as a result of operation:—

1. Female, aged 40 years, with a sphenoidal wing meningioma.

2. Female, aged 67 years, with a suprasellar meningioma.

3. Female, aged 42 years, with a sphenoidal wing meningioma.

All of these patients died within 48 hours from cerebral infarction, and again post-mortem examination was carried out in every case. There is thus no significant difference in mortality between the two groups.

Morbidity.—The morbidity-rate was similarly low in both groups.

A. *Normothermic group*: One case only, a female aged 64 years, with a left pterional meningioma, was re-opened for post-operative clot and then proceeded to make a complete recovery. There were no chest complications of note.

B. *Hypothermic group*: Three cases were re-opened for post-operative clot:—

1. Male, aged 55 years, with an olfactory groove meningioma.

2. Male, aged 58 years, with a cortical meningioma.

3. Female, aged 52 years, with a right parietal parasagittal meningioma.

It is of interest that none of these three cases was acute, one of them being re-opened at twenty-four hours, one on the third post-operative day, and one on the seventh post-operative day, because of unsatisfactory progress. All of these patients subsequently made a good recovery and there is no evidence that the normal processes of tissue hæmostasis are impaired by hypothermia. Neurological recovery was satisfactory in all surviving cases and there was no significant difference to be discovered between the two main groups of patients.

COMMENT

To sum up, it may be said in general that the use of induced hypothermia improves operating conditions, reduces intracranial tension, and limits hæmorrhage and blood transfusion requirements in the majority of cases, but not in all. So far as smoothness of the post-operative course, morbidity, and mortality are concerned, hypothermia confers little, if any, advantage.

BIBLIOGRAPHY

BOTTERELL, E. H., LAUGHHEED, W. M., SCOTT, J. W., and VANDEWATER, S. L. (1956), *J. Neurosurg.*, **13**, 1.

HELLINGS, P. M. (1958), *Brit. med. J.*, **2**, 346.

MCQUISTON, W. O. (1949), *Anesthesiology*, **10**, 590.

ROSOMOFF, H. L., and GILBERT, R. (1955), *Amer. J. Physiol.*, **183**, 19.

ZOLLINGER-ELLISON SYNDROME

REPORT OF A CASE

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CONSIDERABLE interest has been taken recently in the Zollinger-Ellison syndrome which forms a clinical triad characterized by:—

1. Tumour of the cells of the islets of Langerhans in the pancreas.
2. Severe peptic ulceration.
3. Gastric hypersecretion (as much as 2–6 litres overnight containing 50–100 mEq. per litre of free HCl).

Hyperglycæmia is said to stimulate gastric secretion; in the Zollinger-Ellison syndrome, however, hyperglycæmia is neither prominent nor constant, though the adenomata are frequently composed of the alpha cells which secrete glucagon (hyperglycæmic-glycogenolytic factor), and not by the beta cells which secrete insulin.

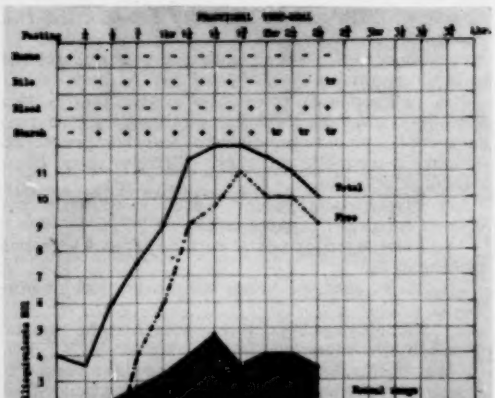

FIG. 192.—Fractional test-meal showing maximum secretion of free HCl to be 11 mEq.

Sufficient cases have now been recorded to make it important to keep pancreatic tumour in mind in all cases of severe or recurrent ulceration. It has been suggested that in such cases it is important to remove the pancreatic tumour and also to do a total gastrectomy.

It has been noted, with regard to this syndrome, that an increasing number of cases are associated with multiple adenomata of the ductless glands, including pituitary, parathyroid, adrenal cortex, and islets of Langerhans.

Though Dragstedt, Montgomery, and Ellis in 1930, and Poth and Fromm in 1950, suggested that the internal secretions of the pancreas may be a factor in the development of peptic ulcer, the association between pancreatic tumours and a severe and progressive type of peptic ulcer was not recognized until Zollinger and Ellison (1955) drew attention to it. Since then at least 80 cases have been described. A review of this syndrome was published in *The Lancet* (1957). Though we here add only 1 case it was thought that the series of investigations justified its publication.

CASE REPORT

Mrs. C. S., aged 46 years, was referred on May 28, 1957, by her family physician, because of pain which she had felt between her shoulders for the preceding six weeks. Two weeks after the onset of the back pain, she felt pain in the epigastrium—spasmodic, and at the peak of intensity causing her to roll about and to get goose flesh. She vomited intermittently, but only a few mouthfuls, and that very bitter. She had lost weight—her best weight ever had been 7 st. 5 lb. She had no family and was separated from her husband.

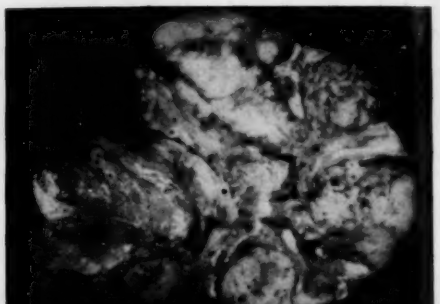

FIG. 193.—The pancreatic tumour as removed at operation (scale in cm.).

On examination she was found to be very thin; she wore an expression of misery and presented the appearance of premature senility. She looked and talked as if she had taken drugs. Her weight was 5 st. 7 lb. The skin was dry and dehydrated. The tongue was moist. The abdomen was distended and bowel movements were visible through the thin abdominal wall. Free fluid was not detected in the peritoneal cavity. No tumour was palpated. The liver was not enlarged. The other systems were normal. The barium meal revealed the presence of a large duodenal ulcer, and on June 24 a two-thirds Polya gastrectomy was performed for a large penetrating ulcer on the posterior wall of the first part of the duodenum. She was allowed home on July 7, though for a few days prior to her discharge she complained of a return of the indigestion pain in the epigastrium. She was readmitted two days later because of the severity of the epigastrium pain. She was interviewed by the psychiatrist, Professor Gibson, who discovered that this patient's mother had died of carcinoma of the stomach, and that the patient had had amenorrhœa

for 2[illegible] years. Gastroscopy was performed by Professor H. W[illegible] Rodgers, who found a hyperactive gastric mucosa. Medi[illegible]al treatment included the use of probanthine.

O[illegible] Oct. 11 she was readmitted with the diagnosis of perfo[illegible]ted ulcer and at operation we found a perforation in an anterior anastomotic ulcer, which was treated by infold[illegible]ng. After a course of intensive medical treatment, a transt[illegible]oracic vagotomy was done by Mr. T. B. Smiley on Nov. 5. Three days later intestinal obstruction (co-incide[illegible]ital) was diagnosed and a transverse colostomy perfo[illegible]med. On March 13, 1958, the colostomy was closed,

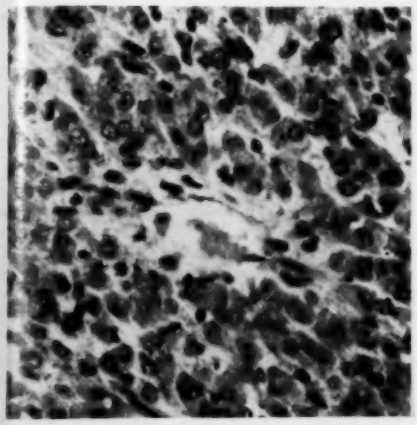

FIG. 194.—Histology of pancreatic tumour—moderately differentiated alpha-cell carcinoma of islet of Langerhans. (× 400.)

and one-third of the stomach remnant was removed. A tumour 7½ cm. in diameter lying above the pancreas and in relation to the left kidney was found. It was first thought to be a tumour of the left suprarenal, but immediately after excision the left suprarenal was found to be in situ, and intact. The spleen and tail of the pancreas were also removed. During and immediately following the operation the patient's condition was well maintained by the transfusion of blood and hydrocortisone. This was stopped after 24 hours. Later the blood-pressure fell to 80 mm. Hg systolic and failed to respond to a noradrenaline drip. The patient died 40 hr. after operation.

The following investigations were carried out during the nine months she was under observation:–

June 6, 1957: Barium meal showed a large penetrating ulcer of the pyloroduodenal region. The antrum was deformed. No delay in emptying was found.

June 11: The white cell-count was 11,500. The serum-amylase was normal. The blood-sugar series: 8 a.m.—93; 12 noon — 95; 5 p.m. — 153; 10 p.m. — 138.

July 17: A barium meal showed evidence of a stomal ulcer, i.e., 23 days after operation of partial gastrectomy. Occult blood test weakly positive.

Nov. 9: Electrolytes Na 141 mEq./l., CO_2 CP29 mEq./l., specific gravity 1024.

Nov. 29: Straight radiograph showed finely mottled calcium deposits on both sides of body of T.12 – ? in pancreas.

Dec. 2: Serum-amylase normal.

Dec. 6: Serum-calcium 12·8. P30. Urea 34 mg. per cent.

Dec. 27: Electrophoresis of large quantity of serum failed to show presence of glucagon.

Jan. 8, 1958: High acid curve in fractional test-meal (*Fig.* 192).

Feb. 13: Serum-calcium 11·3 mg. per cent. Serum-phosphate 2·5 mg. per cent. Clearance P/Ca 24·3 per cent. Urinary calcium 190 mg./24 hr. (Findings consistent with hyperparathyroidism.)

Feb. 14: 17-ketogenic steroids 6·5 mg. 17-ketosteroids 26·9 mg. (Suggestion of overactivity.)

PATHOLOGICAL REPORT.—The surgical material submitted consisted of two portions of stomach, one with a gastro-enterostomy, the pancreatic tumour, and the spleen.

The sleeve of stomach showed a large deep ulcer on its posterior wall adjacent to the pylorus. There was also a shallow ulcer 2 cm. in diameter on the jejunal aspect of the gastro-enterostomy.

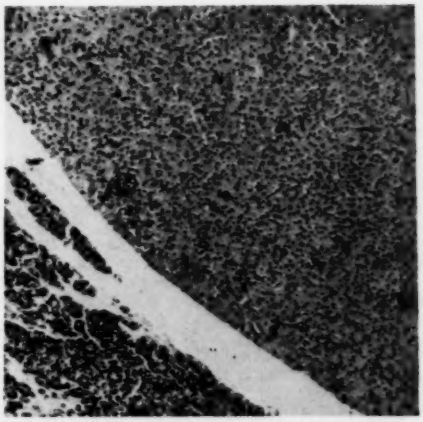

FIG. 195.—Histology of a parathyroid gland—eosinophil adenoma (top right) compressing normal parathyroid tissue below. (× 84.)

The pancreatic tumour (*Fig.* 193) weighed 390 g. and measured 11·5 × 9·5 × 5·5 cm. It appeared encapsulated. On section the tumour was greyish-white and was traversed by strands of fibrous tissue. There were yellowish areas of necrosis.

The spleen appeared normal.

Histology.—The gastric ulcer showed the normal structure of a chronic peptic ulcer which perforated all coats. The stomal ulcer appeared less chronic.

The pancreatic tumour (*Fig.* 194) was very cellular, the cells being spheroidal or oval with a vesicular nucleus. The cells were arranged in sheets or strands and were often separated into alveolar islands by fibrous trabeculæ. Mitotic figures were infrequent and the problem of malignancy would have been difficult if lymphatic channels had not been found to be occupied by tumour cells.

Staining by Gomori's (1941) chrome alum hæmatoxylin and Biggart's (1935) eosin-pyrrol blue methods showed that some of the cells stained as the alpha cells of the islets. No beta cells were identified. Many cells gave a completely negative granular staining.

The diagnosis, therefore, was that of an alpha-cell carcinoma of the islets of Langerhans associated with a chronic peptic ulcer and a stomal ulcer of a gastro-enterostomy.

At autopsy the liver showed numerous secondary deposits, which histologically were of similar nature to that seen in the tumour removed surgically. Only a small percentage of the cells gave a positive granular reaction. The pancreas showed small foci of necrosis which were interpreted as the result of the previous surgery, but histologically several foci of neoplastic proliferation were found. It was impossible to decide whether these represented extension from the primary tumour or were multicentric foci of neoplasia in the islets of Langerhans.

The adrenals weighed 13 g. and showed cortical nodular hyperplasia.

The four parathyroids weighed 0·85 g., and on histological examination were found to show multiple eosinophil-cell adenomata (*Fig.* 195).

The histological examination of the other organs revealed secondary deposits in the lungs, myocardium, and peripancreatic lymph-nodes. The pituitary gland showed no abnormality.

Anatomical Diagnosis.—Islet-cell carcinoma (alpha-cell type) of pancreas, metastases pancreas, regional lymph-nodes, liver, heart, and lungs. Chronic peptic ulcer of stomach. Stomal ulcer of gastro-enterostomy. Multiple eosinophil adenomata parathyroids. Nodular hyperplasia of adrenal cortex.

Biochemical Examination of Pancreatic Tumour.—This patient suffering from fulminant recurrent peptic ulceration was investigated to test the possibility that she might be suffering from a tumour secreting an ulcerogenic agent, possibly glucagon. This was suggested in view of the fact that her blood-sugar curve was of the diabetic type which could be explained as due to a high level of circulating glucagon.

Zollinger and Ellison (1955) observed that the serum of a similar patient of theirs contained a protein fraction separable by electrophoresis, moving more slowly than gamma-globulin and having hyperglycæmic activity. This characteristic of glucagon was confirmed in our case by adding pure glucagon to the patient's serum when, on electrophoresis, a band was observed travelling behind the gamma-globulin. However, the patient's serum when electrophoresized alone showed no such band and it was concluded that by this technique no 'glucagon-like' protein could be found in our patient's serum.

At operation a tumour was removed from the pancreas and was subsequently characterized by the Pathology Department as an alpha-cell tumour of islet tissue. About 2·5 g. of the tumour was received by the Biochemical Department and deep frozen immediately; 1 g. was subsequently removed for analysis and, after thawing, was homogenized in 5 ml. of physiological saline using a Tenbroek tissue grinder. The extract was centrifuged and the supernatant used in subsequent tests for hyperglycæmic and other activity.

Hyperglycæmic activity was assessed by injecting the tissue extract into a dog and carrying out serial estimations of blood-sugar. The results were compared with those obtained when 1 mg. of glucagon in 1 ml. physiological saline was injected into the same dog. The results were as follows:—

Time Schedule	*Blood Glucose using Tissue Extract Injection*	*Blood Glucose using Glucagon Injection*
	per cent	per cent
0 min.	85 mg.	85 mg.
15 min.	74 mg.	115 mg.
30 min.	64 mg.	123 mg.
45 min.	62 mg.	102 mg.
60 min.	72 mg.	83 mg.

From this it is clear that the tissue extract showed no evidence of hyperglycæmic activity such as would be expected were its content of glucagon to be raised.

In the absence of any indication of glucagon in either the serum or the tumour removed from this patient, it was still possible that the tissue extract contained a humoral agent other than glucagon having activity in increasing the gastric secretion. Accordingly, the hyperglycæmic experiment referred to was carried out in a Heidenhain pouch dog and resting juice was collected from the pouch for 1 hour before and after the injection whether of glucagon or tissue extract. In each juice sample the volume *p*H and total titratable acidity were estimated, with the results shown in the following table.

There was thus no evidence that the tumour extract had any action in decreasing the *p*H or increasing the total acid of the juice, nor was there evidence that gl cagon itself had had any significant activity in this respe t. No effect of such a tumour extract on acid secreti n has been reported, and Oberhelman, Nelsen, and Dr gstedt

Juice Sample	Tissue Extract Injection			Glucagon Inj ction		
	Volume	*p*H	mg. Acid	Volume	*p*H	mg. Acid
Resting	0·68 ml.	4·2	0·36	0·84 ml.	4	0·16
1 hr. after injection	1·2 ml.	4·2	0·22	1·2 ml.	3·9	0·18

(1958) found no ulcerogenic activity in an acid-alcohol extract of pancreas obtained from a similar patient.

DISCUSSION

In this case several points are of interest. The history of indigestion extended back for only six weeks prior to her first consultation; the pain was felt in the back for two weeks before it became localized in the epigastrium. From the first it was associated with eructation of mouthfuls of very bitter gastric fluid. The barium showed evidence of a large duodenal ulcer; this was confirmed at operation performed four weeks after first consultation. Her convalescence was rapid and uneventful, except that on the fourteenth day after operation she said "My indigestion pain is back". A barium meal on the twenty-third post-operative day suggested the existence of a stomal ulcer. Following the Polya gastrectomy and vagotomy, her free HCl curve rose to 11 mEq./litre. Various tests suggested that several endocrine glands were showing evidence of abnormal function: the blood-sugar curve was diabetic; the serum-calcium indicated a degree of hyperparathyroidism; the urinary ketosteroids were increased; and the radiograph showed diffuse mottling in the body of the pancreas.

In the largely unknown aetiology of peptic ulcer, as well as the equally unknown reason for the non-digestion of the gastric mucosa by its own secretion, the recognition of the presence of tumour of the alpha cells of the pancreas as a causative factor opens a new and unexpected angle. It is suggested that in cases of severe or recurrent peptic ulcer, the possibility of an alpha-cell tumour of the pancreas should be kept in mind.

We wish to thank Professor J. H. Biggart, Messrs. T. L. Kennedy and T. B. Smiley, and Professor Welbourne for their advice and help.

REFERENCES

Biggart, J. H. (1935), *Edinb. med. J.*, **42**, 42.
Dragstedt, L. R., Montgomery, M. L., and Ellis, J. C. (1930), *Proc. Soc. exp. Biol., N.Y.*, **28**, 109.
Gomori, G. (1941), *Amer. J. Path.*, **17**, 395.
Editorial (1957), *Lancet*, **2**, 1151.
Oberhelman, H. A., Nelsen, T. S., and Dragstedt, L. R. (1958), *Arch. Surg., Chicago*, **77**, 402.
Poth, E. J., and Fromm, S. M. (1950), *Gastroenterology*, **16**, 490.
Zollinger, R. M., and Ellison, E. H. (1955), *Ann. Surg.*, **142**, 709.

PERITONEAL FIBROMATOSIS PRESENTING AS AN ACUTE ABDOMINAL EMERGENCY

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THIS rare disease, even more rarely diagnosed clinically, may remain entirely dormant and unsuspected for many years, and only then reveal its presence by producing marked abdominal symptoms and signs, occasionally of sudden onset. Such a case is here reported.

CASE REPORT

A man, aged 29 years, was admitted to hospital complaining of moderately severe central abdominal colicky pain of 24 hours' duration. He also complained of nausea but had not vomited. There were no other symptoms referable to the alimentary tract or to the urinary system.

FIG. 196.—Section of the fibroma which had undergone torsion. (× 4.)

PREVIOUS MEDICAL HISTORY.—Left inguinal hernia repaired 9 years previously. No previous attack of abdominal pain.

ON EXAMINATION.—A fit man. T. 97°; P. 100. Tongue furred.

Abdomen: Guarding and rebound tenderness over lower abdomen, especially in right iliac fossa. Bowel-sounds normal. No hernia—healed left inguinal scar. P.R. tender in pouch of Douglas.

Total white-cell count—15,000; 92 per cent polymorphonuclear leucocytes.

Acute appendicitis was diagnosed and laparotomy performed.

OPERATION FINDINGS.—Right lower paramedian incision. A little blood-stained free fluid was present. The appendix was found to be normal and was removed. Arising from the peritoneum were numerous pedunculated firm ivory-coloured masses 1·8 cm. in length, some having coarse branches. They were hanging from the stomach, spleen, large and small intestine and their mesenteries, and were most abundant in the pelvis. The liver was unaffected. On the whole these masses were firm, but some were calcified and several free calcified bodies up to 1 cm. in diameter were found in the pelvis. One of these fibrous processes had undergone torsion accounting for the acute symptoms. This was removed and also two others for histological examination. The abdomen was closed without a drain.

The patient made an uneventful recovery. Physical examination and a radiograph of his chest revealed no evidence of tuberculosis. The Mantoux reaction was very weakly positive (1-10,000). He was discharged on the thirteenth day with the wound healed and he was still well when seen one year later.

FIG. 197.—Section showing hyaline connective tissue with active fibroblastic proliferation. (× 136.)

PATHOLOGICAL REPORT.—Polypoid masses 4 cm. × 1 cm., firm and partly calcified (*Fig.* 196).

MICROSCOPICAL EXAMINATION.—These processes are composed largely of hyaline connective tissue with numerous calcified spherules. There are a few areas of active fibroblastic proliferation (*Fig.* 197). The 'polypi' have a mesothelial covering which for the most part consists of a single layer of cells; in some areas, however, there is some proliferation of the mesothelium, but not to such a degree as to suggest that this layer contributes to the tumour formation. One process has strangulated and consists largely of blood-clot with a layer of granulation tissue (epithelial elements are not seen). The lesion appears to be one of peritoneal fibromatosis.

DISCUSSION

Meagre reference is made to benign peritoneal tumours in the text-books of pathology—and only a little more in the literature. They are usually regarded as mesotheliomas, especially in the American literature, although Willis (1948) doubts their existence, believing that they may all be secondary

malignant deposits. However, there appears to be evidence that tumours of this nature can and do occur, although it may be difficult to distinguish between benign and malignant lesions, and their appearance depends on which part of the anatomical peritoneum, mesothelium, or fibrous layer is the site of origin of the tumour. This is, perhaps, best summarized by Thompson (1955) in these words: "The present concept of mesotheliomas emphasizes their pleomorphic nature. While formerly pleural, pericardial, and peritoneal mesotheliomas and solitary fibrous and diffuse superficial mesotheliomas were considered as separate entities, the tendency now is more to consider them merely as pleomorphic forms of the same pathological process. Thus mesotheliomas may be fibroblastic, epithelial, completely undifferentiated, or mixed in type and may involve any serosal surface."

Stout (1954), quoted by Ackerman (1954), classifies these benign tumours as (1) Fibrous—local and diffuse; (2) Papillary—local and diffuse. Out of 56 cases of benign peritoneal and pleural tumours over a period of 32 years, only 2 were fibrous and diffuse and they involved the pleura only. Neither author had seen a case involving the peritoneal cavity.

Clinically, these tumours are silent unless they produce ascites (Rhind and Wright, 1949), or unless one or more processes undergo torsion as in the case here reported. It is possibly never justifiable to diagnose a benign peritoneal tumour clinically, though it may be considered in the differential diagnosis. The true pathological condition more often is revealed by laparotomy for a different lesion or at post-mortem examination.

SUMMARY

A case of peritoneal fibromatosis presenting as an acute abdominal emergency is reported. The pathological and clinical significance is considered.

My thanks are due to Mr. Rose for allowing me to report this case and to Professor Campbell and Dr. Sclare for their report on the histological appearances. I would also like to thank Professor Boyd for his help and advice.

REFERENCES

ACKERMAN, L. V. (1954), *Atlas of Tumor Pathology*, Sect. VI, Fasc. 23 and 24, 97. Washington: Armed Forces Institute of Pathology.

RHIND, J. A., and WRIGHT, C. J. E. (1949), *Brit. J. Surg.*, **36**, 359.

STOUT, A. P. (1954), *Sem. hôp. Paris* (Suppl. Arch. Anat. path.), **30**, 115.

THOMPSON, P. M. (1955), *Amer. J. Surg.*, **90 (b)**, 1007.

WILLIS, R. A. (1948), *Pathology of Tumours*. London: Butterworth.

CHORDOMA OF THE THIRD LUMBAR VERTEBRA SIMULATING CARCINOMA OF THE PROSTATE WITH VERTEBRAL METASTASIS

REPORT OF A CASE

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CHORDOMA is a rare neoplasm of the axial skeleton usually occurring in the sacrococcygeal or basisphenoid regions, but it may be found at any level of the vertebral column.

The following case report illustrates the diagnostic problems presented by a patient with the clinical picture of a cauda equina lesion and a radiograph showing an osteolytic area in the body of the third lumbar vertebra. The correct diagnosis of vertebral chordoma was not made until autopsy, although previous laminectomy and biopsy had been carried out.

CASE REPORT

A male, aged 69 years, a retired Civil Servant, first came under observation during convalescence from a perforated duodenal ulcer which had been successfully operated upon on Dec. 1, 1956. Three days later he developed retention of urine and complained of weakness of both legs.

ON EXAMINATION.—There was facial pallor with evidence of recent weight loss. The bladder was distended and tender. No abnormality of the spine was noted. Rectal examination revealed a small bilobed mobile prostate with no definite evidence of malignancy and there was normal anal sphincter tone.

Neurological examination: The cranial nerves, upper extremities, and trunk were normal.

There were signs of paresis of the lower limbs, affecting the left leg more than the right with absence of tendon-jerks, and flexor plantar responses on both sides. Sensory loss was found in saddle distribution over the sacral and perianal region and this was continuous with a patchy and less definite loss involving both lower limbs.

This was the clinical picture of a cauda equina lesion.

The patient stated that, six months earlier, he had attended another hospital complaining of low back pain and left-sided sciatica of one month's duration. There had been no history of trauma and no difficulty with micturition. Inquiries revealed that a diagnosis of carcinoma of the prostate gland with vertebral metastasis had been made at that time on the evidence of a rather hard but regular prostate felt rectally, a radiograph showing an osteolytic lesion of the body of the third lumbar vertebra, and a total serum acid phosphatase of 4·6 K.-A. units (normal = 0–3 units). Oral stilbœstrol therapy had been prescribed with some relief of symptoms until his emergency admission to St. James' Hospital with a perforated peptic ulcer.

INVESTIGATIONS.—Radiographs of lumbar spine (*Figs.* 198, 199) showed diminution in joint space between the second and third lumbar vertebræ, with secondary osteoarthritic changes suggestive of an old disk lesion. The anteroposterior films showed rarefaction and erosion of the left side of the body of L.3 extending into the lamina and transverse process. Radiographs of pelvis, dorsal spine, chest, and intravenous pyelogram were normal.

The blood picture was normal. Blood-urea: 37 mg. per cent. Serum acid phosphatase: 0·5 K.-A. units per cent. Bence Jones protein was absent from the urine and the serum proteins were normal.

Cystoscopy showed a large middle lobe of prostate and slight bladder trabeculation: no other abnormality was noted. As there was no cystoscopic evidence of a neurogenic bladder, transurethral resection of the bladder neck was performed to relieve obstruction and to obtain biopsy material from the prostate to confirm or disprove the diagnosis of carcinoma. Four and a half grammes of tissue were removed endoscopically and the pathologist reported, "Sections show no evidence of malignancy, but there is little else in the fragments other than fibromuscular tissue."

Lumbar puncture: Clear, colourless fluid obtained. 2–3 white cells per H.P.F.

Protein: 50 mg. per cent.

Sugar and chloride levels were within normal limits and culture gave no growth.

Myelogram: There was a complete block to the passage of the contrast medium in the lumbar subarachnoid space at the level of L.3–4 disk.

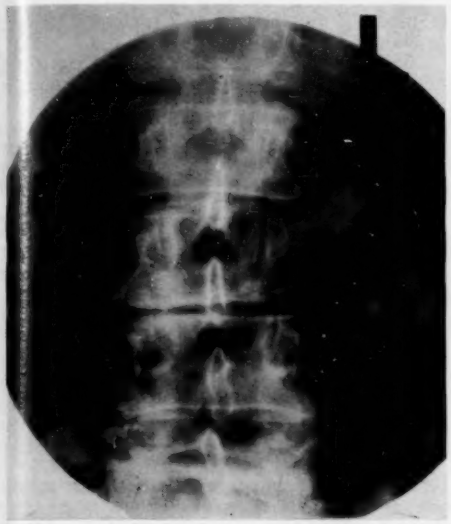

FIG. 198.—Anteroposterior radiograph of lumbar spine, December, 1956, showing erosion and rarefaction of the left side of the body of L.3 vertebra.

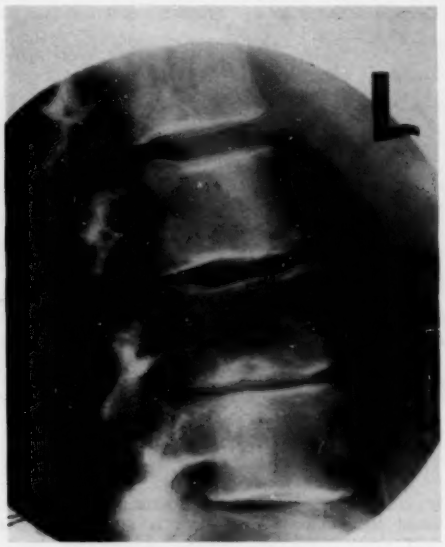

FIG. 199.—Lateral radiograph of lumbar spine, December, 1956, showing rarefaction of the body of L.3 vertebra with narrowing of disk space between L.3 and L.4.

In view of these findings, together with the rapid onset of paraplegia, the patient was transferred to a Neurosurgical Unit for decompression laminectomy. The actual diagnosis remained uncertain, but it was apparent that the cauda equina was compressed at the level of the third lumbar vertebral body and that the most likely cause was a neoplasm, primary or secondary, but a tuberculous lesion could not be excluded.

OPERATION (Jan. 5, 1957).—Through a dorsal midline incision a subperiosteal laminectomy of the third lumbar vertebra was performed without incident. On extending the bone removal laterally to the left, the lower articular facet was found to be unstable and was removed. The third lumbar nerve-root was then identified on the left side and here the dura was adherent and the nerve-root appeared to be expanded. The dura was separated from the underlying ligament laterally, when it was seen that there was a well-defined but rather amorphous membrane anteriorly which was semifluctuant. An incision was made in this avascular membrane and having taken biopsies, a large quantity of rather featureless yellowish, cheesy material was removed from within the cavity of the

FIG. 200.—Photograph of sagittal section of lumbosacral spine removed at autopsy. The whole of the vertebral body of L.3 is destroyed by the chordoma, which is shown compressing the cauda equina posteriorly and invading the adjacent vertebræ.

vertebral body by suction. This achieved adequate decompression and the dural tube pulsated freely following this.

Histology.—The first specimen consists of degenerate ligamentous tissue. Tumour tissue is not present. The second specimen includes some fragments of bone, and

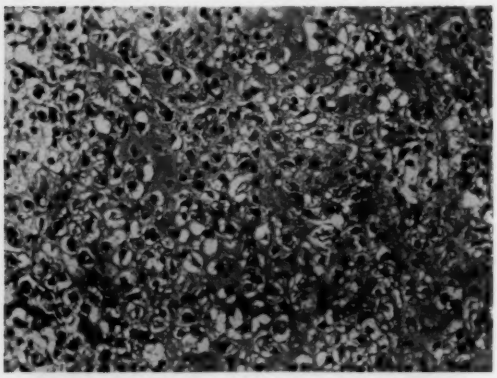

FIG. 201.—Photomicrograph of section of the chordoma, showing the typical large, vacuolated 'physaliperous' cells lying in sheets and clumps in an amorphous matrix. H. and E. (× 150.)

sections made after decalcification show normal bone with attached ligaments. Again there is no tumour tissue present.

PROGRESS.—Following operation, the paraplegia improved and the patient was able to walk with the aid of sticks. But severe back pain developed three months later. This was relieved temporarily by a course of deep X-ray therapy to the lumbar spine, consisting of a dose of 2000 r at 300 kV. over four weeks. Bladder function remained poor and pain recurred after a few months. An autonomous neurogenic bladder, confirmed by repeat cystoscopy, developed and was characterized by intermittent and involuntary micturition. This was controlled to some extent by the use of a penile clamp together with manual compression of the bladder every four hours. However, recurrent attacks of pyelonephritis occurred and the patient finally died in uræmia on March 3, 1958, just two years after the onset of symptoms.

AUTOPSY REPORT.—The relevant findings were:—

1. Bilateral purulent pyelonephritis and cystitis.

2. A white gelatinous tumour was seen invading the psoas muscle mass from both sides of the lower lumbar vertebræ. The body of the third lumbar vertebra was completely destroyed by hæmorrhagic, necrotic, gelatinous tumour which had infiltrated the neighbouring intervertebral disk and surrounding muscle. Posteriorly it had encroached upon the theca, severely compressing the cauda equina, but without evidence of actual macroscopic invasion (*Fig.* 200).

The remainder of the examination was normal.

Histology.—The prostate shows mild benign cystic hyperplasia. The vertebral tumour is composed of large polygonal cells with copious, faintly vacuolated cytoplasm and darkly-staining nuclei. Tumour giant cells with bizarre, hyperchromic nuclei are not infrequent, but mitotic figures could not be found (*Fig.* 201). In some areas these vacuolated cells lie in sheets, with minimal stroma, but in other parts they are dispersed singly or in small clumps in a vast bluish-purple amorphous matrix, into which they appear to merge, in some places, as an ill-defined syncytium engulfed in a mass of vacuolated basophilic material. This material stains metachromatically, is P.A.S.-positive, and stains strongly with Alcian-blue, indicating its acid-mucopolysaccharide nature. The tumour has infiltrated surrounding muscle and connective tissue and in its substance are large areas of hæmorrhage and necrosis. This is a typical chordoma.

DISCUSSION

This case is recorded because of its uncommon site, mode of presentation as a urological problem, and the difficulty with diagnosis.

Chordoma is a rare neoplasm derived from embryonic remnants of the primitive notochord and characterized by slow but invasive growth and a tendency to recur after attempted surgical removal. It very rarely metastasizes and is radio-resistant. Horwitz (1941) pointed out that chordomas arise more often in heterotopic chordal vestiges than in the normal remnant of the notochord, the nucleus pulposus. This would account for the fact that vertebral chordomas appear to arise from the vertebral bodies, as in this patient, rather than the intervertebral disk.

These tumours occur at any age and there is no well-defined peak incidence in any particular decade (Willis, 1953). They occur only in the axial skeleton, and, in a review of 240 cases, Harvey and Dawson (1941) found the following distribution:—

Cranial	37 per cent (88 cases)
Vertebral	12 per cent (30 cases)
Sacrococcygeal	51 per cent (122 cases)

Chordomas arising from the vertebræ are thus uncommon, and Baker and Coley (1953) could find only 47 recorded cases. In a report of this nature it is not intended to review the literature as this has been adequately done from the Mayo Clinic by Dahlin and MacCarty (1952) and Utne and Pugh (1955).

Diagnosis.—The initial presentation of this case with post-operative urinary retention and paraplegia accorded with the diagnosis made elsewhere of carcinoma of the prostate with lumbar vertebral metastasis. As the patient had been on stilbœstrol therapy for the previous six months, rectal examination of the prostate was of no diagnostic aid, and Riches (1949) has pointed out that in prostatic carcinoma the serum acid phosphatase level falls after prolonged stilbœstrol therapy so that this estimation, similarly, was of no help. The radiographic findings of an osteolytic rather than an osteoblastic vertebral lesion, together with cauda equina compression, suggested that the diagnosis might be incorrect and this was further substantiated by the negative prostatic biopsy.

Also osteolytic metastases from carcinoma of the prostate are much less common than osteoblastic lesions (Marks, 1940), and although cord compression associated with osteoblastic secondary vertebral deposits is not infrequent, it is an extreme rarity for an osteolytic metastasis from prostatic carcinoma to cause compression paraplegia. Only 3 such cases were found in a search of the British and American literature over the past thirty years (case records of the Massachusetts General Hospital, 1947; Kawaichi and Rider, 1953; and Carnegie Dickson and Hill, 1933). For these reasons, therefore, the diagnosis of prostatic carcinoma with a secondary vertebral deposit seemed unlikely. In retrospect it is probable that the original urinary retention was due to a neurogenic bladder rather than a middle-lobe obstruction.

Plain radiography of the lumbar spine gave no clue to the correct diagnosis. Utne and Pugh (1955) state that vertebral chordomas present no pathognomonic characteristics on radiographs to differentiate them from other more common spinal tumours. The alternative diagnosis of a staphylococcal or tuberculous vertebral lesion seemed unlikely because of the lack of constitutional symptoms and the absence of local tenderness and muscle spasm. Moreover the radiographs showed involvement of only one vertebral body and no new bone formation. In Pott's disease adjacent vertebral bodies are usually affected, and radiographic evidence of new bone formation is present two or three months after the onset of staphylococcal osteitis.

Lambert Rogers has stressed the importance of laminectomy in these cases of progressive compression of the spinal cord, both as a diagnostic measure and to relieve pain and pressure (Rogers, 1958). In this patient, even after laminectomy and microscopic examination of the tissue removed, diagnosis remained in doubt, but there was gratifying relief of pain and return of function for some months. However, the absence of specific histological features may lessen the value of biopsy in diagnosis as chordomas often contain large hæmorrhagic areas with cystic degeneration and the aggregations of tumour cells may be separated by stromal connective tissues (Willis, 1953). Sissons (1959) describes the typical histological picture as showing large amounts of mucoid intercellular material in which are scattered strands and masses of vacuolated cells. The larger cells are termed 'physalipherous' (bladder-like) because their swollen outlines are indistinct, giving the appearance of a vacuolated syncytium. Willis (1953) states that multinucleated cells are not uncommon while mitotic figures are rare. These points are shown in the photomicrograph (*Fig.* 201) of one of the sections cut from the vertebral tumour.

Treatment.—While radical surgery offers the only hope for ultimate survival in sacrococcygeal chordoma, surgical excision of vertebral lesions has been impossible because of the intimate relationship of the tumour to the spinal cord and nerve-roots. Worthwhile relief from pressure symptoms, however, has been achieved by laminectomy and partial excision.

In the patient who is the subject of this case report, the tumour was irradiated empirically as the diagnosis was not known at the time, and there was some temporary relief of pain. Although chordoma is a relatively radio-resistant tumour, and treatment by irradiation has, on the whole, been disappointing (McWhirter and Dott, 1955), there is a recent encouraging report by Friedman, Hine, and Dresner (1955) describing treatment with supervoltage therapy with relief of symptoms and survival for over three years. As Sir Stanford Cade has stressed in relation to soft-tissue sarcoma (Cade, 1951), 'radioresistance' does not mean that radiotherapy should not be used, nor that many years of survival cannot be achieved by irradiation. The value of roentgen ray therapy is also emphasized by Dahlin and MacCarty (1952). In 2 of their 3 cases of vertebral chordoma treated by radiotherapy alone after biopsy, one had a six-and-a-half year remission of symptoms and the other showed no definite evidence of recurrence five and a half years after treatment. But the literature contains very few reports of survival for long periods. In his authoritative book on bone tumours Coley (1949) reports only 1 case alive more than five years after treatment which consisted of surgery combined with radiotherapy.

SUMMARY

A case of chordoma of the third lumbar vertebra presenting as a cauda equina lesion is recorded.

It was initially diagnosed as carcinoma of the prostate with a solitary metastasis in the lumbar spine.

The diagnosis and the treatment of vertebral chordoma are discussed.

Acknowledgements.—I would like to thank Mr. J. Burke, Consultant Surgeon, St. James' Hospital, Balham, for permission to publish this case which was admitted under his care, and for his helpful advice and criticism.

Dr. M. Israel performed the autopsy and to him I am indebted for the macroscopic post-mortem findings and the histological report.

My thanks are also due to Miss E. Mason for the photographs.

REFERENCES

BAKER, H. W., and COLEY, B. L. (1953), *J. Bone Jt. Surg.*, **35A**, 403.

CADE, S. (1951), *Proc. R. Soc. Med.*, **44**, 19.

Case Records of the Massachusetts General Hospital—Case 33492 (1947), *New Engl. J. Med.*, **237**, 878.

COLEY, B. L. (1949), *Neoplasms of Bone.* New York: Hoebner.

DAHLIN, D. C., and MACCARTY, C. S. (1952), *Cancer*, **5**, 1170.

DICKSON, W. E. C., and HILL, T. R. (1933), *Brit. J. Surg.*, **21**, 677.

FRIEDMAN, M., HINE, G. J., and DRESNER, J. (1955), *Radiology*, **64**, 1.

HARVEY, W. F., and DAWSON, E. K. (1941), *Edinb. med. J.*, **48**, 713.

HORWITZ, T. (1941), *Arch. Path.*, **31**, 354.

KAWAICHI, G. K., and RIDER, R. D. (1953), *J. Urol.*, **70**, 720.

MCWHIRTER, R., and DOTT, N. M. (1955), in *British Practice in Radiotherapy* (ed. Carling, Windeyer, and Smithers). London: Butterworth.

MARKS, J. H. (1940), *Amer. J. Roentgenol.*, **44**, 732.

RICHES, E. W. (1949), *Proc. R. Soc. Med.*, **42**, 481.

ROGERS, LAMBERT (1958), *Brit. J. Surg.*, **45**, 416.

SISSONS, H. A. (1959), in *Modern Trends in Diseases of the Vertebral Column* (ed. Nassim and Burrows). London: Butterworth.

UTNE, J. R., and PUGH, D. G. (1955), *Amer. J. Roentgenol.*, **74**, 593.

WILLIS, R. A. (1953), *Pathology of Tumours*, 2nd ed. London: Butterworth.

BILE PERITONITIS

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BILE peritonitis is an uncommon abdominal catastrophe. However, in spite of its rarity, it forms a subject of considerable interest because of the wide range of pathological processes, some but poorly understood, which give rise to this condition.

In this paper we present a study of the 22 patients with bile peritonitis treated in the last twenty years (1938–58) at the Radcliffe Infirmary, Oxford, together with a review of previous publications on this subject. The important facts concerning the patients in this series are summarized in *Table I*.

Classification.—The causes of bile peritonitis may be classified as follows:—

1. Traumatic rupture of the gall-bladder or bile-ducts by either penetrating wound or closed injury.
2. Post-operative leakage following surgical procedures on the biliary tract.
3. Perforation of the acutely inflamed gall-bladder or duct.
4. Transudation of bile through the walls of a gangrenous but non-perforated gall-bladder.
5. Rupture of a subcapsular cholangitic abscess of the liver in cholangio-hepatitis.
6. Perforation of an apparently normal gall-bladder or duct.
7. Idiopathic, i.e., where there is no demonstrable pathology in the biliary tract.

Two conditions must be differentiated from true bile peritonitis. These are, first, the duodenal contents which escape on perforation of this organ and

Table I.—DETAILS OF 22 PATIENTS WITH BILE PERITONITIS ADMITTED RADCLIFFE INFIRMARY 1938–58

CASE NO.	AETIOLOGY	SEX	AGE	TREATMENT	OUTCOME
1	Traumatic rupture of choledochus cyst	M.	6	Peritoneal drainage	Death
2	Leak from common bile-duct after removal of T-tube following cholecystectomy and exploration of ducts	F.	50	Peritoneal drainage	Recovery
3	Perforation of acutely inflamed gall-bladder	F.	54	Cholecystectomy	Recovery
4		F.	70	Cholecystectomy	Death
5		F.	82	Unfit for any surgery	Death
6		F.	82	Cholecystectomy	Recovery
7		M.	77	Cholecystectomy	Death
8		M.	83	Cholecystectomy	Recovery
9	Perforation of stenosed common bile-duct ? by stone	M.	8 weeks	Choledocho-duodenostomy	Recovery
10	Acute cholecystitis—no perforation	M.	35	Cholecystotomy	Death
11		M.	69	Cholecystectomy	Recovery
12		M.	79	Cholecystotomy	Death
13		M.	88	Cholecystectomy	Recovery
14	Ruptured cholangitic liver abscess—acute cholecystitis	F.	54	Peritoneal drainage	Death
15	Ruptured cholangitic liver abscess—carcinoma of common bile-duct	M.	51	Peritoneal drainage	Death
16	Perforation of normal gall-bladder—hæmorrhagic infarct in thrombocytopenia	M.	51	Cholecystectomy	Recovery
17	Perforation of normal gall-bladder—infarction	F.	62	Cholecystectomy	Recovery
18	Perforation of normal gall-bladder—no cause determined	F.	56	Cholecystotomy	Death (no p.-m.)
19	Perforation of apparently normal common bile-duct	F.	74	Cholecystectomy, drainage common bile-duct	Death
20	Perforation of apparently normal common bile-duct	M.	69	Cholecystotomy, drainage common bile-duct	Death
21	Idiopathic	M.	6 weeks	Laparotomy, exploration common bile-duct	Recovery
22	Idiopathic	F.	62	Laparotomy, cholecysto-jejunostomy	Recovery

which are often heavily bile-stained; and, second, the bilious ascites that occurs in hepatic obstruction.

The sites of leakage in the 22 patients in the present series are summarized in *Table II*.

Table II.—Source of Bile Leakage in 22 Patients with Bile Peritonitis

Organ	Number of Cases	Aetiology
Gall-bladder	13	Perforated acutely inflamed gall-bladder 6 Non-perforated acutely inflamed gall-bladder 4 Perforated macroscopically normal gall-bladder 3
Common bile-duct	5	Traumatic 1 Post-operative 1 Stone in common bile-duct 1 Apparently normal 2
Liver	2	Ruptures of cholangitic abscess
Idiopathic	2	—
	22	

TRAUMATIC RUPTURES OF THE BILIARY SYSTEM

Injury to the gall-bladder by blunt trauma is unusual (Manlove, Quattlebaum, and Ambrus, 1959). The organ may be contused without actual rupture, may be avulsed, or may be lacerated. Occasionally the common bile-duct (Lewis, 1938; Mason, Sidbury, and Guiang, 1954), or the hepatic ducts (Fomon and Hinshaw, 1956), are damaged.

Open injuries, for example gunshot wounds, may occasionally penetrate the gall-bladder but are rarely confined to this organ alone (Gordon-Taylor, 1944).

In this series, there was only one example of trauma to the biliary system and this itself was most unusual:—

Case 1.—**Closed abdominal injury—rupture of choledochus cyst—laparotomy and drainage—death.** A boy, aged 6 years (R.I. 6741), was admitted 48 hr. after having been kicked in the abdomen by a horse. He presented the typical picture of a generalized and advanced peritonitis.

At laparotomy the peritoneal cavity was found to contain copious amounts of bile; no definite source of leakage was found and the abdomen was therefore closed with drainage. A biliary fistula followed, his condition deteriorated, and he died four weeks later.

Autopsy revealed a choledochus cyst of considerable size communicating with the exterior via the fistula. The gall-bladder, cystic and hepatic ducts, and lower end of the common duct were normal. No doubt the collapse of the ruptured cyst had prevented its identification at operation.

Byrne and Bottomley (1953) have reported a case of spontaneous perforation of a choledochus cyst in a 3-day-old infant, but this present example of a traumatic rupture appears unique.

The introduction of needle biopsy of the liver has resulted in a modern cause of traumatic bile peritonitis—at least 2 cases have now been recorded following this procedure (Giraud, Cazal, Latour, and Lévy, 1954; Gallison and Skinner, 1950). It is more likely to occur when the biliary tract is obstructed so that the intrahepatic ducts are widely distended and under pressure.

BILE LEAKAGE FOLLOWING BILIARY TRACT OPERATIONS

Small local collections of bile are common after cholecystectomy, particularly if the common duct is explored and drained—so much so that few surgeons carry out such procedures without routine drainage of Morison's pouch. Fortunately, however, a general biliary peritonitis is unusual. Only 1 case occurred in the present series; this followed removal of a T-tube drain from the common bile-duct. McKenzie (1955) classifies the causes of post-operative bile leakage into five groups: slipping of a ligature on the cystic duct, operative damage to the duct, leakage from accessory ducts in the gall-bladder bed, escape of bile from a dislodged common bile-duct drainage-tube, and seepage through the suture line in the common duct.

He makes the important point that it is often impossible to find the source of leakage at subsequent laparotomy or even autopsy examination; this was so in 12 of his 20 collected cases.

It should also be noted that rupture of the ducts may occur as a late affair weeks or months after cholecystectomy, particularly if the common duct is explored. Newburger (1938) has collected 10 such examples, in 5 of which the stump of the cystic duct had given way; in the remainder a perforation was found in the common duct. In all the cases residual stones were found in the ducts, and he emphasizes that such late perforations are one of the prices to be paid if stones are overlooked at the original operation. Brunschwig (1943) described 2 similar cases; the perforations were found well above the stones, so that they could not be due to simple erosion.

BILE PERITONITIS COMPLICATING ACUTE CHOLECYSTITIS

The acutely inflamed gall-bladder may perforate into the peritoneal cavity either with a resulting generalized biliary peritonitis or with the formation of a localized abscess walled off by omentum. Occasionally perforation occurs into an adjacent viscus (duodenum, transverse colon, liver, or common bile-duct).

Cowley and Harkins (1943) recorded 25 perforations in 2750 gall-bladder operations (0·9 per cent) of which 23 were in 490 operations for acute cholecystitis (4·7 per cent). However, only 6 were free perforations with bile peritonitis. Massie, Coxe, Parker, and Dietrick (1957) found 18 perforations in 1253 cases of cholecystitis (1·4 per cent), but only 6 were unlocalized. Once again the incidence was much higher in acute cholecystitis (4 per cent in 328 acute cases, 0·5 per cent in 925 cases of chronic cholecystitis). Morse, Krynski, and Wright (1957) had a total of 25 perforations in 542 admissions with acute cholecystitis (4·6 per cent) but, of these, only 10 were into the general peritoneal cavity (1·8 per cent). Byrne (1959) noted 5 perforations with general peritonitis in 133 cases of acute cholecystitis (3·7 per cent); 6 others had localized perforations.

In the present series bile peritonitis complicated acute cholecystitis in 11 cases, distributed as follows:—

a. Six patients with perforation of an acutely inflamed gall-bladder (3 deaths).

b. Four patients with gangrene of the gall-bladder without actual perforation but with a generalized bile peritonitis (2 deaths).

c. One patient with rupture of a cholangitic abscess of the liver associated with acute cholecystitis (died).

During the period under review, 795 patients were admitted to the Radcliffe Infirmary with acute cholecystitis, so that the incidence of bile peritonitis

FIG. 202.—Photomicrograph of a section through the site of rupture of a cholangitic liver abscess.

complicating this condition (11 cases) was 1·4 per cent.

Of these 11 patients, 7 were male, 7 were 70 years of age or more (4 were in their eighties), and 8 had one or more stones in the gall-bladder or cystic duct (of which 3 also had stones in the common bile-duct). Seven patients gave no previous history suggesting biliary disease, 3 had had a recent single attack of cholecystitis, and 1 patient had had a series of such attacks in the preceding eight months. Therefore bile peritonitis particularly complicated acute cholecystitis in the elderly male with a short or absent history of previous gall-bladder disease.

It is not proposed to debate the arguments for and against urgent cholecystectomy in acute cholecystitis, but there is one important point we wish to make. It has been stated (Glenn, 1946; Fletcher and Ravdin, 1951) that the risk of perforation of the acutely inflamed gall-bladder is high under conservative treatment and that it is difficult to tell which cases are likely to proceed to gangrene and perforation. In this series of 11 patients, *only* 1 developed this complication while in hospital and this occurred in a cottage hospital on the seventh day of, until then, an undiagnosed illness. The remaining 10 cases were all admitted with an established peritonitis. The incidence of bile peritonitis in acute cholecystitis under conservative treatment while in hospital in this series was therefore only 0·1 per cent, which in itself is hardly a cogent argument for abandoning this method of dealing with the acutely inflamed gall-bladder.

An interesting phenomenon, which is usually overlooked in papers dealing with this subject, is that bile peritonitis may occur when the gall-bladder is wholly or partly gangrenous but not perforated. This occurred in 4 patients in this series.

It is interesting that all 4 examples occurred in male patients, none of whom gave a preceding history suggesting disease of the gall-bladder.

In none of these cases did a careful search of the specimen reveal any perforation, so it is obvious that the gangrenous gall-bladder may become permeable to bile. The available evidence is that it is the mucosa which acts as the barrier layer; Langeron, Langeron, Carlier, and Barbry (1953) have reported a fatal case of bile peritonitis where the gall-bladder was obstructed but not inflamed and where complete desquamation of the mucosa had occurred.

It is not uncommon to find marked local signs of peritonitis in patients with acute cholecystitis being treated conservatively. It may be that here some seepage of bile has occurred into the peritoneal cavity via an area of mucosal necrosis within the acutely inflamed gall-bladder.

BILE PERITONITIS DUE TO RUPTURE OF A CHOLANGITIC ABSCESS

Nauwerk (1905) recorded 2 cases of fatal bile peritonitis each of which, at autopsy, proved to have a minute perforation on the liver surface. In each case there was a calculus impacted at the lower end of the common bile-duct.

Smith (1926) added a further 10 well-recorded cases; all had obstruction of the biliary tree with infection of the ducts. In 7 cases the obstruction was due to a stone and 3 were due to malignant disease. Smith points out that such perforations may be minute, may only be detected by careful search at post-mortem, and perhaps account for some cases of 'idiopathic' bile peritonitis.

In our series 2 examples occurred, one complicating acute cholecystitis, the other in a suppurative cholangitis secondary to a carcinoma of the common duct (*Cases* 14, 15).

The pathological train of events leading up to bile peritonitis in these cases appears to be: obstruction of the biliary ducts due either to stone or neoplasm; infection of the dilated ducts (cholangio-hepatitis); rupture of one or more cholangitic abscesses which lie immediately beneath the liver surface into the peritoneal cavity allowing copious amounts of infected bile, pent up within the distended biliary system, to discharge intraperitoneally (*Fig.* 202).

These abscesses are small and the perforation on to the liver surface may be minute; thus this cause of bile peritonitis may easily be overlooked unless a meticulous search be made.

'SPONTANEOUS' PERFORATION OF THE GALL-BLADDER

The fact that a distended, acutely inflamed gall-bladder may perforate is hardly surprising, but in 3 of the cases in this series the perforated gall-bladder appeared to be quite normal at operation.

Case 16.—**Perforation of the gall-bladder at the site of spontaneous hæmorrhage in thrombocytopenic purpura.**

J. P., male, aged 51 years (R.I. 222850), was admitted with a general peritonitis of 4 hours' duration. He was known to have thrombocytopenic purpura.

At laparotomy the peritoneal cavity was found filled with bile, which was leaking from a small hole in the gall-bladder which was surrounded by a darkened necrotic area about 1 cm. across. Cholecystectomy was performed and the patient made an uneventful recovery.

Histological examination showed that at the margins of the perforation there was complete hæmorrhagic disruption of the entire thickness of the gall-bladder wall. The mucosa elsewhere was very well preserved and the picture was not that of an acute hæmorrhagic cholecystitis.

Case 17.—**Perforation of gall-bladder due to ischæmic infarct.**

G. B., female, aged 62 years (R.I. 128616), was admitted with a general peritonitis of 24 hours' duration.

At laparotomy the peritoneal cavity was full of bile, which was leaking from a minute hole in the fundus of an apparently normal gall-bladder. No stones could be felt either in the gall-bladder or the common duct. Cholecystectomy was performed and recovery was uneventful.

The histological sections of the gall-bladder showed that the wall near the perforation became very much thinner owing to a local defect in the muscularis so that the mucosa and serosa were closely approximated. There was serosal and subserosal necrosis around the puncture, alongside which was a large artery showing an extreme degree of endarteritis.

In these 2 patients there was no obvious reason at operation why their gall-bladders should have perforated. However, a study of the microscopic sections of the gall-bladder wall suggests a possible answer to the problem.

The first patient was known to suffer from thrombocytopenic purpura and a few days before his admission had developed a purpuric rash. Microscopically, the wall of the gall-bladder in the region of the perforation was infiltrated by blood which had disrupted its whole structure. It seems reasonable to assume that this represented an area of spontaneous hæmorrhage which had perforated in its centre.

The outstanding microscopic feature of the gall-bladder in the second case was the atrophy of the walls with complete absence of the muscularis near the perforation. Along with this weakness of the wall there was marked endarteritis in a nearby vessel. When these two facts are considered together it is likely that the perforation was due to the ischæmic infarction of a wall already weakened by atrophy in much the same way that a coronary thrombosis may culminate in rupture of the ventricle. Maingot (1957) had a rather similar patient in whom the perforation was the result of thrombosis of the cystic artery.

In the third patient (*Case* 18) the gall-bladder appeared to be normal at operation but had several yellow subserous patches in the wall, one of which had perforated. No stones could be felt either in the gall-bladder or bile-ducts. Simple cholecystotomy was done but the patient died. There was no autopsy.

IDIOPATHIC BILE PERITONITIS

Occasionally, even on careful search at laparotomy, there is no apparent cause for the biliary peritonitis. Two such examples occurred in this series.

Case 21.—**Idiopathic bile peritonitis in a child.** (This patient has already been fully reported by Davies and Elliot-Smith, 1955.)

P. T., a male child, 6 weeks old (R.I. 81470), was admitted with a 4-week history of obstructive jaundice.

At laparotomy the peritoneal cavity was filled with bile, but no lesion of the biliary system could be detected even on exploration of the common bile-duct. The child recovered.

Case 22.—**Bile peritonitis—no demonstrable cause.**

R. B., a female, aged 62 years (R.I. 101261), gave a typical history of recurrent attacks of cholecystitis for many years.

She was admitted because of 24 hours of severe abdominal pain accompanied by vomiting, and presented the picture of a generalized peritonitis.

At laparotomy a considerable amount of bile was found in the peritoneal cavity. The gall-bladder and ducts appeared to be normal but there was one patch on the cystic duct which was noted to be deeply bile-stained. A cholecysto-jejunostomy was performed with recovery.

Although some authors have postulated theoretical mechanisms of diapedesis or biliary hypertension to explain bile peritonitis with no apparent cause, it is quite inconceivable that bile can leak through the walls of a completely intact biliary system. Leriche (1923) described a case of bile peritonitis in which bile was seen to be oozing through the walls of the gall-bladder "like a veritable spray"; as fast as the beads of bile were wiped away, more oozed on to the surface. This report is often quoted as an example of idiopathic leakage of bile, but in fact the gall-bladder was described as enormous, rigid, and distended; moreover biopsy revealed œdema and round-cell infiltration of its walls.

We have not been able to find an account of this phenomenon of bile 'weeping' through the walls of a gall-bladder which was histologically normal, and we suggest that this occurs only where the gall-bladder is inflamed, where the mucosa has sloughed, and where its impermeability has been lost.

When the biliary system appears intact in the presence of bile peritonitis it is probable that one or other of the following mechanisms is responsible:—

1. Cope (1925) has postulated that a minute perforation of the gall-bladder or biliary ducts occurs either from the ulceration of a small calculus which then becomes lost in the peritoneal cavity, or from inflammation of a mucous gland with subsequent perforation.

Bile escapes into the peritoneal cavity and, with this release of intrabiliary pressure, the gall-bladder and ducts collapse, allowing the tiny perforation to seal off with lymph. Distension of the gall-bladder with further leakage and further sealing may then take place.

Small (1954) reported a patient with bile peritonitis in which he found a patch of discoloration on the gall-bladder wall in the hepatic sulcus. Cholecystectomy was performed and this patch revealed itself as a minute perforation, about a millimetre in diameter, plugged with fibrin. This appears to be an excellent confirmation of Cope's theory and *Case* 22, above, with the patch of bile discoloration over the cystic duct and a previous history strongly suggesting cholecystitis, might also well have had a sealed-off perforation.

2. Perforation of the common duct occurs in its retropancreatic portion with extraperitoneal extravasation of bile and its subsequent leakage into the

peritoneal cavity. Burkitt (1946) described a patient in which this may have taken place and one of Cope's (1925) patients had bile oozing through the posterior peritoneal wall. It is possible that this explanation might account for *Case* 21, where 'idiopathic' bile peritonitis occurred in a child with unexplained obstructive jaundice.

3. A minute perforation of a cholangitic abscess on the liver surface which is overlooked at laparotomy; this has been discussed above.

'SPONTANEOUS' PERFORATION OF THE COMMON BILE-DUCT

Non-traumatic perforation of the extra-hepatic biliary ducts is a rare cause of biliary peritonitis. Hart (1951) was only able to find 10 examples of spontaneous perforation in the literature. Three of these were thought to be due to pressure by a stone, 3 more to intramural infection, 3 to perforation of a diverticulum of the common duct, and the tenth and last to upward traction by a cirrhotic liver.

Gariepy, Capano, and Gardner (1951) believe that erosion by a stone is the commonest cause of perforations of the bile-ducts, but suggest that other possible reasons may be obstruction of the duct, active cholangitis with necrosis of the wall, thrombosis of vessels, and reflux of pancreatic juice.

In the present series of 22 cases 3 were found at operation to have a perforation of the common bile-duct as a cause of the biliary peritonitis. An eight-week-old infant (*Case* 9) had a complete stenosis at the lower end of the common bile-duct, and alongside the perforation was a small pigmented stone. Davies and Elliot-Smith (1955), when they reported this case, thought that the obstruction was almost certainly the cause of the perforation. They did, however, concede that erosion by the stone was a possibility.

In the other two cases (*Cases* 19, 20) no stones were found either in the gall-bladder or in the ducts, nor was there any obstruction.

Thrombosis of one of the vessels supplying the common bile-duct leading to infarction and subsequent perforation of the wall is an attractive theory. Moreover, as has already been discussed, this sequence of events was almost certainly the underlying pathological process in one of the perforated gall-bladders described (*Case* 17). There seems to be considerable disagreement about the adequacy or otherwise of the blood-supply to the common duct. Shapiro and Robillard (1946) claimed that the arterial supply is meagre, and obviously if this is so a clot in what virtually amounts to an end-artery might easily culminate in a perforation. In contradiction to this work is the study of Douglass and Cutter (1948), who found that in 84 per cent of cases there was an abundant anastomotic blood-supply to the ducts which made perforation by infarction highly improbable. Henley (1955) concludes that most of the blood-supply to the common duct came from a single vessel called the 'retroduodenal artery studies Wilkie', and it appears from his injection of that the supraduodenal part of the duct where the perforations are usually found has a relatively poor blood-supply. However, Hart (1951) seems to be the only author who is prepared to accept a thrombosis of the mural arteries as a cause for the perforation; certainly in the 3 cases we report there was nothing to suggest that the hole found at operation was due to an infarct of the duct wall.

A more likely explanation is that the perforation is the result of a mural infection of the bile-duct akin to that found in the gall-bladder in acute cholecystitis. Burden (1925) has shown that the mucous membrane of the common bile-duct is studded with glands which in many instances almost completely penetrate the wall. He has also demonstrated that in cholecystitis there is almost invariably an accompanying inflammation of the bile-ducts with distension of these glands by mucus. It seems reasonable to assume that in some cases the inflammation so weakens the wall that it ruptures in exactly the same way as an acutely inflamed gall-bladder or appendix may eventually perforate. In *Cases* 19 and 20 an acutely inflamed gall-bladder was found at operation, so that if Burden's view is correct the ducts were probably also inflamed and thus more liable to perforate.

The fourth and last possibility to be considered is that the perforation is due to reflux of pancreatic juice. There seems little to recommend it beyond the fact that traditionally it finds a place in the list.

SUMMARY OF CLINICAL PICTURE

In this series of 22 patients there were 13 males and 9 females, with ages ranging from 6 weeks to 88 years. Eleven of the patients died.

All, except 2 infants, presented with acute abdominal pain, in some starting and staying on the right side of the abdomen, in others commencing centrally and moving to the right, and, in a few, being generalized from its first appearance. In a few cases the pain appeared with dramatic suddenness and intensity so that within a few hours they were in hospital. Most of the patients, however, had a longer history (thirty hours on the average), the first pain being comparatively slight but then getting progressively worse and finally becoming severe. Mild vomiting occurred in most cases, but only 1 patient was jaundiced. Cope (1925) claims that the one constant feature of the condition is a remission of the pain after the first attack, but this did not occur in the present series. Although the one traumatic case in this series had the same symptoms and signs as the other patients, many of the reports (Lewis, 1938; Smith and Hastings, 1954; Mason, Sidbury, and Guiang, 1954) on such patients suggest that they run a slower and more benign course rather like that of bile peritonitis in infancy.

The two infants with biliary peritonitis provide a bewildering and startling contrast to the adults. Instead of being gravely ill, both were in reasonably good health. Jaundice and abdominal distension lasting for several weeks replaced the symptoms of a few hours of severe abdominal pain. Their abdomens were soft, with nothing to suggest a peritonitis in place of the tenderness and rigidity found in the adults. Both survived, yet more than half the adults died. That these two cases are not exceptional is borne out by the study of Gertz (1959), who found that in 9 further cases of bile peritonitis in childhood

6 had clinical findings substantially as those described above. One is, therefore, driven to the paradoxical conclusion that while in adults the natural history of the disease is short and likely to end fatally, in infants it is much slower, with recovery as the most likely outcome.

Mortality.—Why there is such a high mortality in adults has been one of the most persistent puzzles of biliary peritonitis. McWilliams (1912) reported a mortality of 48 per cent in 108 patients, and just over forty years later, with all the aids and advances of modern surgery, half the patients in the present series died. In discussions on this point three possible theories are put forward to account for the high mortality (Douglas and Grey Turner, 1940; Bell and Warden, 1955; Aird, 1957):—

1. The bile is infected and produces a bacterial peritonitis (Miles and Jack, 1953).

2. Excessive fluid loss into the peritoneal cavity results in severe shock (Harkins, Harmon, and Hudson, 1936).

3. Cholæmia results from absorption of bile-salts (Wangensteen, 1926).

One would expect that if the escaping bile is infected, the condition becomes more serious and the patient is more likely to die. On the other hand, if the bile is sterile, as in trauma and possibly in infants, the resulting peritonitis is chemical, and provided the initial extravasation is not large, the irritant effects of the bile-salts can be considerably modified by the dilution of the peritoneal exudate. Excessive fluid loss into the peritoneum leading to a state of irreversible shock is another possibility, but Miles and Jack (1953) calculated that the amount of peritoneal exudate was too small to account for death in the majority of cases. Wangensteen (1926), as the result of injecting bile intraperitoneally into dogs, concluded that cholæmia from the absorption of bile-salts was the cause of death, but Rewbridge (1931) in similar experiments was unable to detect any rise in the blood-level of the bile-salts.

In deciding whether a patient with general peritonitis lives or dies, age and length of history are of supreme importance. The older the patient the worse the outlook, and the longer the history the more likely is death. Excluding the 2 infants, the average age of the 9 patients who lived was 63 years and of the 11 who died, 59 years—no great difference. However, there is a marked disparity in the length of the history of the two groups—27 hours in the survivors as against 48 hours in those who died. When one considers that in perforated peptic ulcers the mortality may rise to 60 per cent when operation is delayed for 36 hours (Maingot, 1955), it is not surprising that biliary peritonitis kills half of its victims.

SUMMARY

A series of 22 patients with bile peritonitis is presented and the subject reviewed. The aetiology of many such cases is readily explained, but some examples occur as a result of perforation of an apparently normal gall-bladder or duct; in others, there is no demonstrable pathology in the biliary tract. Possible reasons to account for these phenomena are discussed.

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REFERENCES

Aird, I. (1957), *Companion in Surgical Studies*, 963. Edinburgh: Livingstone.
Bell, L. G., and Warden, H. D. (1955), *Amer. J. Surg.*, **89**, 579.
Brunschwig, A. (1943), *Surgery*, **13**, 951.
Burden, V. G. (1925), *Ann. Surg.*, **82**, 584.
Burkitt, D. P. (1946), *Brit. med. J.*, **2**, 155.
Byrne, J. J., and Bottomley, G. T. (1953), *Amer. J. Dis. Child.*, **85**, 694.
— — (1959), *Amer. J. Surg.*, **97**, 156.
Cope, Z. (1925), *Brit. J. Surg.*, **13**, 120.
Cowley, L. L., and Harkins, H. N. (1943), *Surg. Gynec. Obstet.*, **77**, 661.
Davies, P. A., and Elliot-Smith, A. (1955), *Arch. Dis. Childh.*, **30**, 174.
Douglas, D. M., and Turner, G. Grey (1940), *Brit. med. J.*, **2**, 280.
Douglass, T. C., and Cutter, W. W. (1948), *Arch. Surg., Chicago*, **57**, 599.
Fletcher, A. G., jun., and Ravdin, I. S. (1951), *Amer. J. Surg.*, **81**, 178.
Fomon, J. J., and Hinshaw, J. R. (1956), *Surgery*, **39**, 322.
Gallison, D. T., and Skinner, D. (1950), *New Engl. J. Med.*, **243**, 47.
Gariepy, L. J., Capano, O. A., and Gardner, L. Wm. (1951), *Amer. J. Surg.*, **81**, 357.
Gertz, T. C. (1959), *Arch. Surg., Chicago*, **78**, 7.
Giraud, G., Cazal, P., Latour, H., and Lévy, A. (1954), *Pr. méd.*, **62**, 445.
Glenn, F. (1946), *Surg. Gynec. Obstet.*, **83**, 50.
Gordon-Taylor, G. (1944), *Brit. J. Surg.*, **32**, 247.
Harkins, H. N., Harmon, P. H., and Hudson, J. E. (1936), *J. Amer. med. Ass.*, **107**, 948.
Hart, D. E. (1951), *Ann. Surg.*, **133**, 280.
Henley, F. A. (1955), *Brit. J. Surg.*, **43**, 75.
Langeron, L., Langeron, P., Carlier, G., and Barbry, A. (1953), *Pr. méd.*, **61**, 1789.
Leriche, R. (1923), *Ibid.*, **31**, 252.
Lewis, K. M. (1938), *Ann. Surg.*, **108**, 237.
McKenzie, G. (1955), *Aust. N.Z. J. Surg.*, **24**, 181.
McWilliams, C. A. (1912), *Ann. Surg.*, **55**, 245.
Maingot, R. (1955), *Abdominal Operations*, 305. New York: Appleton.
— — (1957), *The Management of Abdominal Operations*, 827. London: H. K. Lewis.
Manlove, C. H., Quattlebaum, F. W., and Ambrus, L. (1959), *Amer. J. Surg.*, **97**, 113.
Mason, L. B., Sidbury, J. B., and Guiang, S. (1954), *Ann. Surg.*, **140**, 234.
Massie, J. R., Coxe, III, J. W., Parker, C., and Dietrick, R. (1957), *Ibid.*, **145**, 825.
Miles, R. M., and Jack, H. S. (1953), *Surgery*, **34**, 445.
Morse, L., Krynski, B., and Wright, A. R. (1957), *Amer. J. Surg.*, **94**, 772.
Nauwerk, C. (1905), *Münch. med. Wschr.*, **52**, 931.
Newburger, B. (1938), *Ann. Surg.*, **107**, 558.
Rewbridge, A. G. (1931), *Surg. Gynec. Obstet.*, **52**, 205.
Shapiro, A. L., and Robillard, G. L. (1946), *Arch. Surg., Chicago*, **52**, 571.
Small, W. P. (1954), *Brit. J. Surg.*, **41**, 552.
Smith, S. W., and Hastings, T. N. (1954), *Ann. Surg.*, **139**, 517.
Smith, W. (1926), *Ibid.*, **83**, 55.
Wangensteen, O. H. (1926), *Ibid.*, **84**, 691.

ANTERIOR SPINAL FUSION

THE OPERATIVE APPROACH AND PATHOLOGICAL FINDINGS IN 412 PATIENTS WITH POTT'S DISEASE OF THE SPINE

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In 1956 two of the authors (Hodgson and Stock) published a preliminary report on the first 48 patients with Pott's disease submitted by them to the operation of anterior spinal fusion. The object of this operation was to allow full visualization of the lesion by a wide anterior exposure to facilitate complete excision of all diseased and devitalized tissue, and to provide for immediate fusion by means of a strut graft in compression between adjacent healthy vertebral bodies. The authors' hopes that the attainment of all three objectives would result in greatly accelerated healing and a reduction in the period of hospitalization and incapacity have been well justified. The first 48 patients have been followed for 4 years and the results of treatment of the first 100 consecutive cases reviewed two or more years after operation have been reported elsewhere (Hodgson and Stock, 1960). They show a fusion rate of 93 per cent, a mortality of 4 per cent, complete recovery from paraplegia in 26 out of 35 cases (74 per cent), and partial recovery in a further 6 cases. There was one relapse of the disease within the period of follow-up. The patient was re-explored anteriorly, a residual abscess evacuated, and re-grafting carried out. Sound healing has occurred.

The purpose of the present communication is to review the operative approaches used at all levels of the spine in a series of 412 patients and to report on both the operative findings and immediate mortality and complications in this series. Much of the pathology seen has seemed to us strange, for it is not reported in any of the present-day literature, but on pursuing our search further we have found that many of our findings have been reported in the last century.

Anterior exposure of the spine has also been found useful in cases of prolapsed intervertebral disk, secondary carcinoma in the spine, spinal biopsies, spondylolisthesis, and other conditions, but although the methods of approach are identical, it is not intended to refer to the operative findings in this paper.

THE SURGICAL APPROACH

Fortunately the majority of cases of tuberculosis of the spine involve the middle and lower thoracic vertebræ. This is the area which is most readily displayed anteriorly through a standard thoracotomy and the operative procedure in this area will be described first. Many of the details of exposure and fixation are applicable to other areas of the spine, and in the subsequent description of the procedures at other levels we shall refer principally to the differences between them and the standard thoracotomy approach.

Isolated instances of a transthoracic approach have been described by Nissen and Nigst (1956) and by Cauchoix (1956), the latter in a personal communication to one of us.

Mid Thoracic (T.4–T.11).—The chest is usually opened on the left side, where the aorta forms a useful landmark. On the right side the inferior vena cava may become incorporated with the abscess wall and is liable to be injured. In cases where there is a very large abscess on the right side with nothing on the left, or when the abscess appears to be separating from the spine or when there is some pulmonary complication such as abscess or empyema, an approach may be made through a right thoracotomy.

An incision is made along the rib which, in the mid-axillary line, lies opposite the point of maximum convexity of the kyphus. This is usually two ribs higher than the centre of the vertebral lesion. It is desirable to enter the chest near the upper border of the disease process, for owing to the slope of the ribs it has been found easier to work downwards beneath the lower ribs than upwards beneath the upper ones. The abscess is usually seen at once, but there may be adhesions between it and the adjacent lung which require separation. The lung must be mobilized and pushed forwards. A vertical incision is made in the groove between aorta and abscess. It is important that this incision be made close to the aorta, for if made more laterally the pleura is densely adherent to abscess wall. The aorta is pushed forwards and medially, displaying the intercostal vessels, which must be secured and divided between ligatures or after diathermy coagulation for the whole length of the abscess cavity. This usually requires the division of four or five vessels, but in patients with very early disease it may be less. We have seen no evidence that division of any number of vessels on one side impairs the blood-supply to the spinal cord even when the important 10th intercostal artery is divided. The maximum number of vessels divided in any case was eight. During the division of these arteries, the hemiazygos vein and intercostal veins are seen, and the latter together with elements of the splanchnic nerves require division. When division of vessels is complete the aorta can be displaced forwards away from the spine and the abscess palpated across the front of the vertebral bodies.

The abscess is opened by a T-shaped incision, made first transversely opposite the centre of the disease process in the spine and completed by a vertical incision medial to the distally placed ligatures on intercostal vessels. By this means the blood-supply to the abscess wall is very largely controlled and two triangular flaps can be raised, displaying the diseased area including the inside of the abscess cavity. Pus, debris, and sequestrated bone or disk are evacuated by suction or with pituitary rongeurs. At this stage it is usually possible to pass the sucker right across in front of or between diseased bodies into the abscess cavity on the right side and evacuate all material from there also.

The next step is to remove diseased bone which has not become sequestrated and which may be dense, sclerotic, and avascular, or soft and friable due to active disease. There is relatively little bleeding at this stage and an attempt must be made not to enter normal vascular bone until all diseased tissue has been removed. This is not difficult, for the disease is usually limited at each end by a normal disk which is naturally avascular. All diseased bone whether soft or sclerotic is excised with osteotome, chisel, or rongeurs so that the spinal canal is exposed for the whole length of the disease. The posterior common ligament is often destroyed. A layer of granulation tissue forms around the anterior group of extradural veins and gradually becomes organized into a thick fibrous layer. In our early cases we mistook this for the posterior common ligament, but repeated histological examination has shown its true identity. This fibrous layer must be removed with knife or rongeurs. The dura covering the spinal cord is now exposed and in almost all cases is clean, shining, and pulsating. It is such a common occurrence to find collections of pus or sequestrated bone or disk in the spinal canal behind relatively normal posterior parts of diseased bodies even in patients without paraplegia, that we regard it as imperative to excise the whole of every vertebral body affected by the disease, and to excise the posterior common ligament or the tuberculous granulation tissue and fibrous tissue behind it to inspect the dura. If the dura is not pulsating, if there is severe indentation, or if the paraplegia is of long standing, it is desirable to open the dura and inspect the cord, but this should not be done without a definite indication as we have had 4 cases with a fistula developing between subarachnoid space and pleural cavity, in one instance leading to a fatal tuberculous meningitis.

When the whole of this procedure has been completed the disk at each end of the cavity is removed, exposing normal bleeding bone, and one or more strut grafts fashioned from the rib removed during the initial thoracotomy are inserted. These may be supplemented if necessary with frozen banked rib grafts. In the lumbar spine we would normally use an iliac crest graft as this is more massive, fuses more quickly, and is readily available near the operation site.

Streptomycin (1–2 g.) and isoniazid (100–200 mg.) are placed in the cavity and the pleura resutured. It is only in this part of the abscess wall that there is any remaining evidence of tuberculous disease, and we have no evidence that this cannot be controlled adequately by systemic and local chemotherapy once the main disease process is excised.

The chest is closed in the usual way with suction drainage of the pleural space for two or three days.

Upper Cervical (C.1, 2).—Tuberculous disease of the spine in the first two cervical vertebræ is relatively uncommon and we have not been able to devise an entirely satisfactory approach to these vertebral bodies. A high lateral approach similar to that used in the middle cervical region gives very limited access, and although we have been able to reach an abscess for drainage we were not able to expose the bodies adequately by this route for full débridement and fusion. Two of us have used a transoral approach to the anterior arch of the first cervical vertebra and the odontoid process of the second in 4 cases of atlanto-axial dislocation, and have used a similar approach in 2 cases of Pott's disease. A tracheotomy is performed to ensure a good airway throughout the operation and post-operative period. With the mouth open, the soft palate is turned forwards and held in place with a stitch. A vertical incision is made in the midline of the posterior pharyngeal wall and the edges retracted and sutured laterally. The anterior surface of the bodies is displayed as soon as the abscess is opened. Débridement and grafting can be carried out without difficulty. An anterior approach has also been developed through the thyrohyoid membrane to reach the body of the second and third cervical vertebræ and has been used in 7 cases of tuberculosis. Both of these approaches will be described elsewhere (Fang and Ong). The oral approach has been used by Southwick and Robinson (1957) for non-tuberculous conditions, while the subhyoid approach was originally described by Langenbeck (Kocher, 1911) for the treatment of benign and malignant tumours of the larynx.

The upper cervical spine is very difficult of access and it remains to be seen which approach will prove to be the most satisfactory. At the present time, however, the transoral and transthyrohyoid routes seem to offer good prospects. They are not without risk and careful attention must be paid to the accurate apposition of the posterior pharyngeal wall, to hæmostasis, and to the prevention and management of respiratory and feeding difficulties in the post-operative period. One patient died as a result of aspiration of food into the larynx 6 weeks after operation, when phonation and respiration were normal.

Lower Cervical (C.3–7).—The vertebræ from the third to the seventh cervical present no serious problems of access, and the approach was described by the two senior authors in 1956. An anterior approach has also been used by Southwick and Robinson (1957), Cloward (1958), Bailey and Badgley (1960), principally for non-tuberculous conditions.

The collar incision gives limited but adequate access in most cases, but if necessary an oblique incision along one or other border of the sternomastoid provides more room. The incision may be on either side of the neck, but the side of the abscess should be chosen. The sternomastoid may be retracted or if necessary divided, the carotid sheath, pharynx, larynx, trachea, and œsophagus are retracted, and the front of the cervical bodies displayed, covered by prevertebral fascia, and the anterior common ligament, which is incorporated in the wall of the abscess cavity. The abscess cavity is incised vertically, i.e., parallel to the spine. In doing this it may be necessary to incise some fibres of the scalene muscles, but the whole procedure is relatively avascular.

Upper Thoracic (C.7–T.3).—Next to the first and second cervical vertebræ this is one of the most difficult areas to approach adequately. There are two standard approaches: In our first report we favoured one through the right third rib with the scapula displaced upwards and forwards. This provides good exposure of the abscess, which can be

incised and evacuated. The bodies tend to collapse together but can be separated without difficulty and diseased tissue can be safely excised with chisel or rongeurs. The lowest part of the brachial plexus is seen in this approach but does not interfere with the evacuation of diseased tissue. The principal difficulty lies not with the exposure but with the subsequent fusion, for the spine at this area is reversing its normal thoracic curvature and the bodies are relatively small. In addition there is always an element of scoliosis and rotation in these cases and it is not easy to maintain adequate fixation of the bone-graft by compression.

Some six months after publication of our first report, at a joint meeting of the Royal College of Surgeons of England and the Académie de Chirurgie de Paris held in London, Cauchoix and Binet (1957) described an alternative anterior approach to this area of the spine by a sternal split. Through a vertical midline incision the sternum is divided from the suprasternal notch to the xyphoid. The thymus is retracted, the innominate vein divided, the trachea and œsophagus are displaced to the left, and the abscess is revealed. It is, however, at the bottom of a rather deep dark hole in which blood collects. The bodies tend to collapse together and are less easy to separate than in the approach described above. Excision of bone with a chisel must be done with extreme care, for not only must cuts be made directly backwards towards the spinal cord with risk of damage by the osteotome, but every blow will transmit pressure to the cord through any sequestra or disks which may lie in the spinal canal. Finally bone-grafting is even more difficult through the approach than through a third-rib thoracotomy. We have used the trans-sternal Cauchoix approach in 10 cases, but feel that it is unnecessarily difficult and access is limited by the large number of important structures which are liable to damage. Four cases (40 per cent) have died, which compares very unfavourably with the mortality of less than 4 per cent in the entire series.

For these reasons we feel that the third-rib approach is preferable.

Lumbar Spine.—When disease involves T.12 and L.1 and 2 the most satisfactory approach is through an eleventh-rib incision described by Fey (Mouat, 1939). This can be kept extrapleural and retroperitoneal and the diaphragm can be separated from the vertebral body. The upper part of psoas is divided transversely and the muscle turned downward. Lumbar arteries and veins are more difficult to control than the intercostal vessels, as it is impossible to secure them in the para-aortic groove, but there is otherwise no essential difference from the thoracic operation.

In disease of the lower lumbar spine a renal approach may be made using the twelfth-rib incision described by Digby (1941) and others. In this case it is usually necessary to divide the psoas transversely lower down and go through an ill-defined abscess between muscle-fibres. Care must be taken not to damage the trunks of the lumbar plexus which lie behind the posterior parts of that muscle.

Lumbosacral Spine.—Disease affecting the fifth lumbar and first sacral vertebræ may be approached through a low renal type incision commencing at the tip of the twelfth rib, but access is far from satisfactory. The iliac vessels lie in front of the fifth lumbar and are easily damaged, and for that reason when disease is restricted to the fourth and fifth lumbar and first sacral vertebræ we have used a purely anterior transperitoneal approach. The patient is placed in the Trendelenburg position and the abdomen opened through a lower midline incision. The viscera are packed off in the upper abdomen. The peritoneum is incised below the bifurcation of the aorta and the abscess opened. Care must be taken to avoid injury to the left iliac vein which crosses the upper part of the fifth lumbar vertebra, but should it be damaged it can be sutured readily. As in the case of the trans-sternal approach to the upper thoracic spine, care must be taken to avoid displacing fragments of bone backwards or effecting nerve damage by the chisel, but fortunately here the spinal canal is wide and only the remains of the cauda equina is present, which is less susceptible to damage. Furthermore the exposure is much

Table I.—Analysis of Approaches

Level	Approach	Series A (Hodgson, Stock, Fang)	Series B (Stock, Fang, Ong)	Total
Cervical 1–3	Lateral	1	0	1
	Transoral	0	2	2
	Thyrohyoid	0	7	7
Cervical 2–7	Lateral	5	3	8
Upper thoracic C.7–D.3	Thoracic R.	3	2	10
	Thoracic L.	10	2	12
	Trans-sternal	5	5	10
Thoracic D.4–L.2	Right	13	1	14
	Left	148	45	193
Thoracolumbar D.12–L.2	Fey	29	7	36
Lumbar L.1–5	Renal	67	30	97
Lumbosacral	Renal	5	1	6
	Transperitoneal	7	13	20
Sacral	Perineal	1	1	2
		299 (294 patients)	119 (118 patients)	418 (412 patients)

wider and visualization better than with the transsternal approach. The excisional procedure is similar to that described earlier and grafting is usually done with an iliac-crest graft.

Up to March, 1960, 412 patients have been subjected to anterior fusion involving 418 operations (6 patients having disease at more than one level). The distribution of disease and methods of approach are summarized in *Table I*.

PATHOLOGICAL FINDINGS

1. The Vertebral Bodies.—We have found that the disease in the spine is almost always more extensive than the X-ray picture suggests. The normal appearance of the disease is erosion near the intervertebral disk with separation of the disk. Farther away, but still within the same vertebra, may be found small pockets of caseous material usually extending right up to the next disk. Small fragments of bone between these pockets undergo avascular necrosis and become sequestra separating from adjacent bone and prolapsing laterally into the abscess cavity or backwards into the spinal canal though usually remaining anterior to the thick fibrous layer which has formed around the anterior extradural veins. These fragments vary in size and rarely exceed 5 mm. in their longest axis, but on occasions much larger prolapsed fragments may be found, particularly when the whole of the posterior part of the vertebral body has become necrotic. In some cases, and this is especially true in the lower thoracic and lumbar spine, a large segment of a vertebral body may become avascular without completely separating from normal bone. During operation this is easily recognized as dense bone which bleeds very little.

The diseased bodies collapse as the necrotic bone is displaced laterally and posteriorly, but the spine often remains mobile even in advanced disease and can be straightened by direct pressure posteriorly over the kyphus. This manœuvre assists in the débridement and in the evacuation of the abscess cavity on the side opposite to that of the approach.

On occasions the vertebral body may be distended by caseous material within a thin shell and this is more likely to be seen in patients who have been confined to bed for this disease without any active antibiotic treatment. This is the 'dactylitic' type and can usually, but not invariably, be recognized radiologically. It is this type which may collapse to form a 'vertebra plana', a thin layer of dense bone formed by the upper and lower parts of the collapsed body coming together.

In many chronic cases healing commences, but is usually incomplete. Fresh vascular bone may be found surrounding pockets of pus, and in some of these cases there may be a posterior bony spur or ridge which indents the common ligament as well as the cord and its coverings. In all cases the posterior parts of the vertebral bodies must be removed so that the spinal canal can be opened for the whole length of the disease and any such ridges removed.

2. The Disk.—It is a radiological characteristic of tuberculosis of the spine that the disk space is diminished. In most cases this is not due to destruction of the disk by the tuberculous process, for it is remarkably resistant to infection. In the early stages it is probably due to interference with nutrition and consequent dehydration of the disk. Later it is due to separation of the disk from its adjacent vertebræ and its prolapse into some part of the abscess cavity. Like bony sequestra, the disk may be found in the spinal canal and pressure from it can cause paraplegia. Not infrequently the disk is eroded, but it is probable that this is due to pressure from irregular rough vertebral bodies, for histologically the disk shows little evidence of infection except at the periphery.

3. The Meninges and Spinal Cord.—The posterior common ligament of the spine may form a posterior wall of the abscess cavity. It is, however, very thin and is usually destroyed. A thick layer of fibrous tissue and granulation tissue is often present, lightly adherent to the dura, from which it can be stripped without undue difficulty. On occasions, however, the dura may be firmly adherent and rarely the tuberculous process may involve the dura, pia-arachnoid, and even the spinal cord. Frank meningitis is extremely uncommon, although 2 cases of tuberculous meningitis have followed anterior decompression in our series of 418 operations, one as a direct result of the operation.

In cases of chronic disease, and especially in cases of paraplegia, it is a commonplace to find a thick transverse ridge of fibrous tissue pressing on the dura and causing indentation or even acute angulation of the spinal cord with localized ischæmia. Such thickened ridges must be removed.

If there is serious indentation of the dura, if pulsation fails to return, or if the paraplegia is of long standing, it is wise to open the dura to inspect the spinal cord. In most cases this appears normal, but in long-standing paraplegia there may be great narrowing of the cord, which in extreme cases we have seen reduced to a few shreds of nerve tissue. In such cases there is little prospect at all of recovery of function and it is desirable that the surgeon should know this from the beginning.

4. The Lung and Pleural Cavity.—In about half our cases of tuberculous disease of the thoracic spine with a paravertebral abscess we have found adhesions between the lung and the abscess. In the majority of cases the adjacent lung is normal or, at most, showing areas of patchy collapse and the adhesion thin and filmy. In these cases there is no evidence to support the contention (Burke, 1940, 1948) that the disease spreads from lung to spinal column. After division of the adhesions the lung is fully mobilized and can usually be inflated to its full extent by the anæsthetist. In a smaller group of cases the adhesions may be thick and vascular and the visceral pleura closely adherent over a wider area to the abscess wall. In a smaller number still (25) we have found penetration of the paravertebral abscess across adherent pleura into lung tissue to form a localized pulmonary abscess surrounded by atelectatic lung. On separation of the lung from the paravertebral abscess, caseous material can be evacuated from the pulmonary abscess and when this has been done there is rarely any air leak which would suggest communication with a bronchus. It has sufficed in all cases to insufflate the cavity with streptomycin and to close the cavity from within by suture. In 2 cases we have found that the pulmonary abscess has separated itself from the paravertebral one, forming two separated abscesses though linked by

adhesions. In both cases the spine showed evidence of incomplete healing of the disease.

In 4 cases the paravertebral abscess had penetrated the pleural cavity but not the lung, forming a chronic empyema with grossly thickened pleura. In 2 cases an opening in the empyema cavity led directly to the diseased spine. In all these cases, in addition to the routine excisional procedure on the spine, it was necessary to perform decortication of the lung and chest wall. All cases of pulmonary abscess and empyema which have been followed for sufficiently long periods have showed healing of the spine without any complication attributable to the additional procedures necessary.

Penetration of the lung was recognized many years ago and Galen shrewdly remarked that when the curvature was high up in the spine evacuation of matter usually took place through the lungs. In the nineteenth century there were numerous case reports of this character where paravertebral abscesses ruptured into the lung and bronchus with caseous material and even sequestrated fragments of bone being coughed up.

5. Aorta.—In all cases when the spine is collapsed, the configuration of the aorta is more or less distorted. In the majority of cases the aorta kinks to the left but its lumen is not interrupted. In none of the cases in this series has the aorta been adherent to the abscess wall and after division of intercostal vessels it has been possible to lift it forwards with ease.

In 2 cases, however, which have not been included in this series, there has been penetration of the aorta in association with a destructive lesion in the spine suspected of being tuberculous. Both were explored and found to be inoperable as fluid blood surrounded the spine. The first patient, a male aged 48 years, had a fracture of the spine many years previously. Much later he developed pain, kyphosis, and paraplegia. After an unsuccessful attempt at anterior fusion, the damaged segment of aorta was resected and replaced with a dacron graft. The patient did not survive and post-mortem examination was refused. The second patient, a female aged 27 years, was first explored posteriorly and fused. A small quantity of pus of tuberculous type was found adjacent to a lamina. A subsequent attempt at anterior fusion was abandoned.

These cases have not been included in the series because histological, bacteriological, and visual evidence of the tuberculous origin of the disease is missing. Nevertheless caseous material was found posteriorly in the second case and it is probable that at least one, if not both, of these cases represent penetration of the aorta by a tuberculous spinal abscess.*

Other instances have been reported by Dewes (1852) and Fuller (1859) and more recently by Somerville and Wishart (1948), Simpson and Grobbelaar (1955), and Kovalenko (1959) of patients in which the abscess had become adherent to and penetrated the aorta with either massive dissemination of the disease or rapid death from a rupture of the aorta.

6. Œsophagus.—In none of our cases has the œsophagus been involved in the abscess wall and we have seen no cases of fistula formation following penetration of the œsophagus. Nevertheless Johnson (1923) reported a case of cervical caries which penetrated the œsophagus in which the fistula was closed and the abscess subsequently drained with satisfactory results. Brand (1939) reported perforation of a traction diverticulum of the œsophagus in a patient who had a tuberculous empyema secondary to thoracic spinal caries.

7. Other Organs.—We have seen 1 instance of penetration of the colon by the abscess. Penetration of the duodenum has been reported by Davies Colley (1886); of the bladder by G. Buckstone Browne (1891); of the vena cava by Butcher (1857); and of the liver, colon, and rectum by Kovalenko (1959).

MORTALITY AND COMPLICATIONS

The operation can be one of considerable severity and should never be undertaken lightly. It should not be undertaken at all unless there are adequate facilities for pre-operative investigation and preparation, satisfactory anæsthesia, and good post-operative care for a patient who is to have a thoracotomy. Nevertheless the operation is not of equal severity in all patients and it is safest in young patients with very limited disease. All our deaths have occurred in patients with extensive bony disease or with spread elsewhere in the body, but not all were directly attributable to the operative procedure.

No case was refused operation by the authors because of the general condition of the patient, but in a few instances operation has been delayed on account of miliary tuberculosis or severe pulmonary involvement.

In this series of 412 patients 18 have died since the operation was performed. Of these, however, 1 died 3 months after her spinal fusion as a result of hepatic failure due to cholangiohepatitis which had been treated by cholecystectomy. One died from aspiration of food 6 weeks after a thyrohyoid approach. The patient was being fed by the mother contrary to instructions with the child in the head-down, hyperextended position. Four more died of tuberculosis, 3 or more months after the spinal fusion and cannot be regarded as part of the operative mortality. Twelve died within 3 months of the operation. The operative mortality is therefore 12 out of 412 or 2·9 per cent, while the total mortality from the disease in the period under review is 16 out of 412, or 3·9 per cent (*Table II*).

Although this mortality is reasonably low, nevertheless one cannot look at the causes of death in these cases without realizing that some might have been avoided. Over-transfusion with saline and hæmorrhage into a tracheotomy tube might have been avoided, and atelectasis due to bronchial obstruction with mucus may fall within the same category. The remaining operative deaths, however, were due almost entirely to the advanced state of the disease when the operation was undertaken.

No deaths occurred in any patients in whom the disease was of limited extent, of short duration, and in whom there was no pulmonary involvement.

Complications are numerous, but not severe, and have been reviewed elsewhere (Hodgson and Stock, 1960). One complication which has been encountered

* Tubercle bacilli have been since cultured from the caseous material in the second case.

four times following excision of part of the dura and in one case preceded a fatal termination is the formation of a cerebrospinal fistula into the pleural cavity. In the cases which ended in death there had been quadriplegia of many years' duration.

Table II.—MORTALITY

CASE NO.	SEX	AGE	APPROACH	TIME SINCE OPERATION	REMARKS
1. Operative Deaths.—					
A. 42	M.	9	Thoracotomy	1 dy.	Very extensive spinal disease 6 yr. duration. Over-transfusion with saline
A. 60	M.	4	Thoracotomy	7 dy.	Paraplegia. Bilateral pulmonary disease. Broncho-pneumonia
A. 82	F.	6	Thoracotomy	4 hr.	Paraplegia. Very extensive disease C.5–D.7. Very shocked
A.108	M.	10	Thoracotomy	2 mth.	Left atelectasis. Pleural effusion. Right broncho-pneumonia. ? C.S.F. Fistula to pleura
A.111	M.	2	Thoracotomy	3 mth.	Miliary tuberculosis. Tb. meningitis. ? C.S.F. Fistula to pleura
A.117	M.	55	Thoracotomy	4 hr.	Paraplegia. Respiratory obstruction ? due to mucus
A.153	M.	3	Trans-sternal	3 dy.	Tracheobronchitis and atelectasis due to mucus
A.221	M.	6	Trans-sternal	4 hr.	Hæmorrhage into tracheotomy tube
A.267	F.	23	Cervical	6 dy.	Quadriplegia. Bilateral pulmonary tuberculosis
B. 16	F.	4	Thoracotomy	4 hr.	Subarachnoid hæmorrhage. ? cause
B. 45	M.	2	Thoracotomy	3 hr.	Extensive pulmonary disease. Atelectasis
B. 72	F.	5	Trans-sternal	3 hr.	Atelectasis. Tracheotomy. Hæmorrhage into tracheo-tomy tube
2. Deaths due to Primary Disease.—					
A.119	F.	37	Thoracotomy	3 mth.	Developed tuberculous meningitis
A.162	F.	7	Trans-sternal	3 mth.	Miliary tuberculosis. Paraplegia
A.279	M.	51	Thoracotomy	3 mth.	Paraplegia. Extensive pulmonary disease. Tb. broncho-pneumonia
B. 30	M.	45	Thoracotomy	6 mth.	Paraplegia. Extensive pulmonary disease. Pyelo-nephritis
3. Deaths unrelated to Tuberculosis.—					
A. 87	F.	67			Developed acute cholecystitis and cholangitis 3 mth. after satisfactory anterior fusion. Died of hepatic failure after cholecystectomy
B. 77	M.	3	Transthyro-hyoid	6 wk.	Aspiration of food

Acute dilatation of the stomach has been seen in a few cases involving the thoracolumbar spine. Convulsions have occurred in 2 patients with cervico-dorsal disease.

There have been no instances of tuberculous infection of a previously clean pleural cavity or of tuberculous wound infection. There have been a few instances of wound infection, usually in association with a braided non-absorbable suture.

Four patients in whom decortication was carried out for a tuberculous empyema at the same time as the spinal fusion have shown satisfactory re-expansion of the lung.

CONCLUSIONS

Anterior spinal fusion was devised as a method of direct attack on the disease focus facilitating complete excision of the focus and immediate grafting by a strut in compression. It has proved possible to approach all levels of the spine from the front to carry out these objectives, and, fortunately, that area of the spine which is most frequently involved is also the easiest to approach.

The operation, although of some magnitude, does not carry a high mortality-rate except in the trans-sternal approach. Indeed in early cases with limited spinal lesion and no disease elsewhere, the operative mortality approaches zero. We feel therefore that the operation should be advised in all early cases of tuberculous spinal disease as being the quickest and most certain way of ensuring sound healing. It should supplant completely conservative treatment in most cases. It is only in advanced cases that there should be any hesitation in advocating operative interference, although we ourselves have not refused to operate on a single case.

The anterior approach has demonstrated that the disease frequently is more extensive than was suspected clinically or radiologically, and by revealing small foci in adjacent vertebræ has indicated the cause for recurrence of the disease at either end of an area of spine which has been fused posteriorly. The direct approach and treatment of pleural or pulmonary extension improves pulmonary function and lessens the risk of relapse later.

The results of anterior spinal fusion reported in detail elsewhere show a fusion rate of 93 per cent even when judged by the most stringent clinical and radiological criteria, and this in conjunction with the low mortality-rate indicates that the method represents a significant advance in the treatment of spinal tuberculosis.

REFERENCES

BAILEY, R. W., and BADGLEY, C. E. (1960), *J. Bone Jt Surg.*, **42A**, 565.

BRAND, M. A. (1939), *Amer. Rev. Tuberc.*, **40**, 473.

BROWNE, G. B. (1891), *Trans. clin. Soc. Lond.*, **24**, 58.

BURKE, H. E. (1940), *J. thorac Surg.*, **9**, 506.

— — (1948), *Trans. Amer. Clin. Climat. Ass.*, **59**, 122.

BUTCHER, R. G. H. (1857), *Dublin Quart. J. med. Sci.*, **21B**, 271.

CAUCHOIX, J. (1956), personal communication.

— — and BINET, J. P. (1957), *Ann. R. Coll. Surg. Engl.*, **21**, 237.

CLOWARD, R. B. (1958), *J. Neurosurg.*, **15**, 602.

DAVIES COLLEY, J. N. C. (1886), *Tr. path. Soc. Lond.*, **37**, 561.

DEWES, E. (1852), *Lond. J. Med.*, **4**, 35.
DIGBY, K. H. (1941), *Surg. Gynec. Obstet.*, **73**, 84.
FANG, H. S. Y., and ONG, G. B., "The Anterior Approach to the Upper Cervical Vertebræ" (in preparation).
FEY, B. (1925–27), *Arch. urol.*, **5**, 169 (quoted by MOUAT).
FULLER, H. W. (1859), *Brit. med. J.*, **2**, 537.
HODGSON, A. R., and STOCK, F. E. (1956), *Brit. J. Surg.*, **44**, 266.
— — — — (1960), *J. Bone Jt Surg.*, **42A**, 295.
JOHNSON, J. (1923), *Minnesota Med.*, **6**, 56.
KOCHER, T. (1911), *Operative Surgery*, 3rd English ed., p. 434. London: Black.
KOVALENKO, D. G. (1959), *Orthop. Travm. I. Protez.*, **20**, 21 (Russian text abstracted in *Excerpta Medica* (Surgery), 1959, **13**, 1029).
MOUAT, T. B. (1939), *Brit. J. Urol.*, **11**, 126.
NISSEN, R., and NIGST, H. (1956), *Helv. Chir. acta*, **23**, 329.
SIMPSON, T. V., and GROBBELAAR, B. G. (1955), *J. Bone Jt Surg.*, **37B**, 614.
SOMERVILLE, G. W., and WISHART, J. (1948), *Ibid.*, **30B**, 327.
SOUTHWICK, W. O., and ROBINSON, R. A. (1957), *Ibid.*, **39A**, 631.

NON-SPECIFIC MESENTERIC ADENITIS

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NON-SPECIFIC mesenteric adenitis is now well recognized as a common cause of acute and chronic abdominal pain in children. Enlargement of the mesenteric glands can occur in many different diseases, but the cause of non-specific mesenteric adenitis is not known; it is in fact possible that there is more than one cause. The clinical picture and operative findings are quite definite, and there seems to be sufficient justification for regarding it as a clinical entity. This paper presents a series of 49 children—aged between 2 and 13—with mesenteric adenitis who have been under my own observation, together with a long-term follow-up. Of the 49, 27 had the diagnosis confirmed by operation; in the other 22 it was felt that a diagnosis could be made on clinical grounds with reasonable confidence. The two groups are considered separately, because only the operative group consists of proved cases, and also it was felt that the follow-up would be more informative if the two could be compared.

REVIEW OF THE LITERATURE

Tuberculous mesenteric adenitis, or tabes mesenterica, has been recognized for over two hundred years. The first description of non-tuberculous adenitis was by Wilensky (1920), who described 3 cases of mesenteric adenitis; 1 was tuberculous, 1 suppurative, and 1 a typical non-specific mesenteric adenitis. He pointed out that some intra-abdominal abscesses were due to suppurative adenitis. Brennemann (1921) noted the occurrence of abdominal pain in children with sore throats. Mesenteric adenitis was found at operation in 2 of his patients, and he thought that this was the cause of the pain. Struthers, also in 1921, described a series of 22 cases of mesenteric adenitis, most of which, but not all, were thought to be tuberculous. Of the 4 case reports, one is very suggestive of non-specific adenitis. Heusser (1923), quoted by Wilensky and Hahn (1926), stated that there was no basis for regarding the condition as tuberculous after examining a number of excised glands and failing to isolate tubercle bacilli. Freeman (1923) described the appearance at operation and distinguished these cases from tuberculosis. In a later paper (1929) he thought that the majority of diseased glands were definitely not tuberculous, as tabes mesenterica was rare and the condition described was common. He assumed that the disease was self-limiting, and that it rarely developed into a frank tabes mesenterica. Since then, numerous papers have been published, but the disease has only recently been mentioned in the latest text-books.

PATHOLOGY

The appearances at operation are quite characteristic. There may be a small serous peritoneal effusion. The glands are discretely enlarged, firm, mobile, and greyish-white in colour, with an almost translucent appearance. The juxta-intestinal glands are always smaller than the central ones; it is impossible to see the glands at the root of the mesentery through the usual gridiron incision, but they can be palpated and are often large. The glands are usually confined to the last few feet of the ileum, but they may extend to the jejunum. In one case there were enlarged glands in the mesocolon and the porta hepatis. The only other reference in the literature to extramesenteric glands is by Fitzsimons (1946), who found glands in the mesocolon, near the pylorus and duodenum, and around the aorta at the level of the diaphragm. He suggested that they may be caused by a low-grade peritonitis. The glands never show any evidence of tuberculosis or pyogenic infection. One child seen while this survey was being conducted had a very large gland in the ileocæcal region which was removed. On section it showed acute pyogenic infection. Cases of inflammation proceeding to suppuration have been reported (Wilensky, 1920; Strang, 1957), but there is no reason for regarding these as non-specific mesenteric adenitis, and this case has accordingly not been included.

The appearance of the appendix varies. In the present series the appendix was normal in 13 cases, acutely inflamed in 3 cases, and chronically inflamed in the remaining 11 cases. Foster (1938, 1939) stated that the appendix was involved in 90 per cent of cases, and that the disease was associated with chronic appendicitis. Other authors disagree; Postelthwait and Campbell (1949), Rosenburg (1937), and Ward-McQuaid (1951) found that the appendix was

grossly normal in almost all cases. Madigan and Coffey (1950) reported no marked abnormality in the histology of the appendix apart from some cases where there was an acute appendicitis. They pointed out that there was sometimes a lymphoid hyperplasia in the appendix, and swelling of the lymphoid tissue could cause an acute appendicitis.

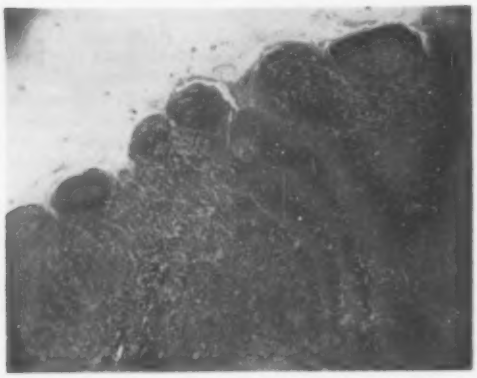

FIG. 203.—Low-power view of lymph-gland showing non-specific reactive hyperplasia. H. and E. (× 20.)

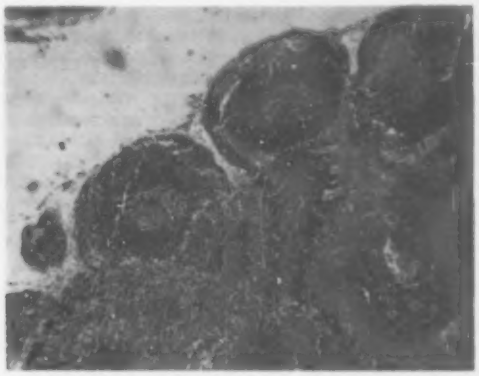

FIG. 204.—High-power view of lymph follicles with Fleming's centres. H. and E. (× 47.)

The terminal ileum is nearly always normal. Aird (1945), however, suggests that a mild non-specific regional ileitis may be present at the onset of the disease. Some authors postulate that toxins may be absorbed through a normal bowel, but this has never been proved. Almost all writers agree that diarrhœa is an exceptional symptom, and this fact is against an ileitis as a factor in the aetiology.

Many workers have removed glands for histological examination, but no specific changes have ever been found; the picture is that of a simple reactive hyperplasia.

In this series glands were not removed for histological examination. A biopsy is, of course, performed if there is any evidence of specific infection, or if the glandular enlargement is suspected to be secondary to a condition such as glandular fever or a blood dyscrasia.

Reports are available in three cases of non-specific mesenteric adenitis from a later series; they are not included here because of the short follow-up.

The typical appearances are shown in *Figs.* 203, 204. The lymph-follicles are enlarged and the germinal centres are hyperplastic; these enlarged prominent areas are sometimes known as 'Fleming's centres'. There is proliferation of the reticulum cells of the sinusoids, which are often dilated and contain lymphocytes. This picture represents a non-specific reaction of the lymph-gland to some irritant or infection.

Bacteriological investigation is usually negative, but occasionally normal intestinal bacteria have been isolated, mostly enterococci and *Bact. coli*. Goldberg and Nathanson (1934) succeeded in culturing hæmolytic streptococci from the throats of all their patients, but only in 1 case from the glands. Ireland (1938) isolated a hæmolytic streptococcus in 1 patient who had a suppurative mesenteric adenitis. Studies of the antistreptolysin O titre were estimated in 50 cases by Fitzgerald (1956), and he concluded that hæmolytic streptococcal infection was not a factor. Gage (1939) cultured an enterococcus in 94 per cent of cases, but thought that it was not the cause of the adenitis as the agglutination tests were negative.

The possibility of a virus infection has been suggested (White and Collins, 1936; Fitzgerald, 1956), and in favour of this is the association with upper respiratory infection and the complete absence of chronic inflammatory changes in the glands (Freeman, 1929). Webster and Madore (1949) carried out experimental inoculations of animals with a filtrate from the glands and the results were all negative. Raftery, Trafas, and McClure (1950) found histoplasmosis in 43 per cent of their cases, and Collins (1955) concluded that histoplasmosis was the main cause of mesenteric adenitis. Their work has not been confirmed in this country, and further investigation is required on this point.

The question of parasitic infection has also never been proved; if threadworms are present, they are probably coincidental. Threadworms are present in 10 per cent of otherwise normal appendices, and they are sometimes a cause of abdominal pain in children and may be associated with chronic appendicitis.

The social distribution of mesenteric adenitis has been studied by some workers. On the whole, there have been no significant results, and my own experience supports this view. Two papers are of particular interest here: Asencio (1949) reported that the disease was common in Puerto Rico where conditions were very poor, and Fitzsimons's (1946) patients were often underweight, underdeveloped, and anæmic. Fitzsimons thought that this was the result of the disease, and that it was an important cause of underdevelopment in children. These findings are in contrast to the majority of authors, who find that most of the children are physically normal. It lends weight to the suggestion that there may be more than one cause of mesenteric adenitis.

The occurrence of upper respiratory tract infection and its bearing on the aetiology have aroused

considerable interest. Among other authors, Wilensky and Hahn (1926) draw an analogy between the cervical glands in tonsillar infection and the mesenteric glands. Upper respiratory infection is so common in children that a definite relationship is difficult to prove. Estimates in reported cases vary from the view of Klein (1938), who stated that upper respiratory tract infection was not a factor, to a 55 per cent incidence (Postelthwait and Campbell, 1949) and Goldberg and Nathanson's series (1934), where all their patients had a streptococcal throat infection and a clinical incidence of 70 per cent of upper respiratory infection. In the present series there was a history of sore throats, colds, or bronchitis in 30 per cent of the operative group and 45 per cent of the non-operative group. The patients who complained of sore throats with abdominal pain had both symptoms relieved by operation. One child who was regarded as having a typical mesenteric adenitis had no further symptoms after tonsillectomy.

CLINICAL

In this series mesenteric adenitis has presented frequently in a chronic form. The majority of references in the literature have been to acute mesenteric adenitis, and one of the purposes of this paper is to emphasize the chronic aspect of the disease. Nineteen patients who had an appendicectomy were admitted to hospital as acute abdominal emergencies, but, of these, 10 had a definite history of previous attacks of pain. This history can often be elicited from the parents after operation, but is easily missed unless specifically sought for.

The main symptom is pain, and the chief characteristic of the pain is that it is intermittent, colicky, or spasmodic. It comes on suddenly, lasts a few minutes, and quickly settles. It may be quite severe, and is usually accompanied by pallor. In between spasms the patient is fairly comfortable. The site of the pain may vary from time to time, and may be difficult to localize. It is usually a mid-abdominal colic, but is sometimes in the right iliac fossa (*Table I*). Vomiting may or may not be present, but bowel disturbances are the exception rather than the rule. In the present series constipation was present in 7 cases and diarrhœa in 5 cases. In most of the previously reported cases diarrhœa was uncommon. There was slight urinary frequency in 3 patients.

Table I.—SYMPTOMS OF 49 PATIENTS DIAGNOSED AS MESENTERIC ADENITIS

Symptoms	*Operative Cases*	*Treated Medically*
Pain:		
Central abdomen	13	15
Right iliac fossa	6	4
Both sides	7	3
Vomiting	13	11
Headache	13	6
Pallor	13	10

Headache occurs in abdominal migraine and in the periodic syndrome, but it has not been noted as a common symptom in the previously reported cases of mesenteric adenitis. Thirteen patients in the operative group and 5 in the conservative group complained of headache; in 3 cases the pain was suggestive of migraine. This point is discussed later.

On examination there is almost always some abdominal tenderness in the right iliac fossa, or below and to the right of the umbilicus. The tenderness is not well localized, and, like the pain, it may vary in position at different examinations. Shifting tenderness, as described by Klein (1938), has not been found to be a useful diagnostic sign as it is rather difficult to elicit satisfactorily. Abdominal rigidity is never present in the chronic cases, and only rarely in the acute episodes. This finding agrees with Donhauser (1957) but differs from the older accounts. In this series the children have never been really ill and physical signs have not been well marked. Palpation of the enlarged glands as described by Fitzgerald (1956) has also been found difficult to elicit with confidence, although the glands can certainly be felt in some cases.

Several authors have described acute and fulminating varieties of mesenteric adenitis (Rosenburg, 1937; Klein, 1938). The fulminating type has not been encountered. There appears to be no difference in the appearances at operation between chronic, acute, and fulminating cases, and no explanation has been offered as to why these patients should be so ill. They seem to recover rapidly after operation.

All authors agree that there is no laboratory investigation which will confirm a diagnosis of mesenteric adenitis; they may, however, be required to exclude other diseases. A full blood-count has been done in almost all cases. Most patients have had a normal blood-count, only 1 had a leucocytosis of over 20,000, and 6 had a leucocytosis of over 12,000. The leucocytosis bore no relation to the acuteness of the symptoms, but the acute cases tended to show a shift to the left. A relative lymphocytosis was noted in 5 of the operative cases and 8 of the non-operative group. Five patients had a monocytosis of over 10 per cent.

DIFFERENTIAL DIAGNOSIS

The mesenteric glands may be affected primarily by any disease of the reticulo-endothelial system. They may also be enlarged secondarily in dysentery, Crohn's disease, the exanthemata, and rheumatic fever. Ward-McQuaid (1951) reported 3 cases of dysentery, 2 cases of scarlet fever, and 1 case each of hepatitis, measles, and glandular fever, which developed whilst the patient was under observation for mesenteric adenitis. Goldberg and Nathanson (1934) had 1 patient who developed pericarditis after operation and which progressed to a typical rheumatic fever. Fitzgerald (1956) noted the occurrence of a mesenteric syndrome in rheumatic fever. Two children, not included in this report, had an appendicectomy in the prodromal stage of infective hepatitis; a well-marked mesenteric adenitis was found in both these patients. The first essential in diagnosis is to exclude these specific diseases in which the glands are enlarged secondarily.

As seen above, mesenteric adenitis can be acute, and there may be acute exacerbations of a chronic state. In this case the most important differential diagnosis is acute appendicitis. This has been well covered by Aird (1945) and it is possible in practice to distinguish between them with a fair degree of accuracy. In difficult cases the advice of Gross (1953) can be followed and the patient observed for an hour

or two. In mesenteric adenitis the pain tends to settle quickly. If an acute appendicitis cannot then be excluded, operation must be advised.

The differential diagnosis of the chronic case is a difficult problem; again appendicitis is a common cause of pain, and its importance is well realized. Very little attention has been directed to the periodic syndrome and abdominal migraine, but it is most important that they should be recognized although their relationship to mesenteric adenitis is not clear.

Abdominal migraine, although the name has been criticized as contradictory, is simply migraine with abdominal symptoms. Farquhar (1956) reported a series of children with recurrent headache, abdominal pain, and vomiting, either singly or in combination, with a history of migraine in one or both parents. He laid down the diagnostic criteria for migraine, and thought that cyclical vomiting could be regarded as the same disease. All patients should be questioned about headaches and the family history should be investigated. It should be remembered that migraine may present as abdominal pain in children, and as the child grows older, the abdominal pain is replaced by headache. Some parents have given a history of migraine and stated that they had had abdominal pain as children. If there is a suspicion of migraine, the child should, of course, be treated medically. Three cases in this series are of particular interest. On being questioned in the follow-up clinic one of them gave a clear history of headache and a fortification spectrum. After appendicectomy all his symptoms were abolished; at the present date, four years after operation, he has no headaches but complains of slight occasional pain. Another patient had a mesenteric adenitis proved by operation; five years later he still has attacks of abdominal pain and vomiting with headaches, and there is a family history of migraine. A history of migraine was obtained in the follow-up in one girl in the conservatively treated group.

The periodic syndrome (Reimann, 1957; *British Medical Journal*, 1957) is probably a number of disorders which are grouped for convenience under this heading. It can be defined as a disease of infants and children, sometimes affecting several members of a family, and at times hereditary, and which consists of acute paroxysmal episodes showing a certain periodicity. The symptoms may be mainly articular, abdominal, or febrile. Kempton (1956) stresses the usually regular periodicity of the disease, the main features of which are abdominal pain, headache, pallor, vomiting, and pyrexia. There is usually a nervous element and often an upper respiratory infection acting as a trigger. This could well be a description of mesenteric adenitis, and Kempton regards mesenteric adenitis as only one aspect of the periodic syndrome. The relationship between periodic disease and migraine has been discussed by Hamilton (1951).

The diagnosis between mesenteric adenitis and the periodic syndrome is therefore difficult. A history of periodicity is the crucial point in diagnosis, and also a family history of similar attacks or of cyclical vomiting.

Chronic appendicitis can usually be distinguished as the symptoms and signs are rather better defined. The pain is less spasmodic, the attacks last longer, and the child may be ill for a day or two. Between attacks he is more liable to be off colour, with some general malaise. On examination the tenderness is greater and more localized. Tuberculosis, either peritoneal or mesenteric, becomes more obvious after a period of observation. The fibrocaseous type now seems to be rare. Although tuberculous cervical glands are fairly common, tabes mesenterica was not seen while this survey was being conducted. Other diseases can usually be excluded after a period of observation. Two conditions deserve special mention. An eosinophilia in the full blood-count should be taken as an indication to search for an allergic basis for the symptoms. Renal disease is not common, but it is important that it should be recognized, and an intravenous pyelogram should be done to exclude it in doubtful cases.

TREATMENT

It is often not possible to make a firm diagnosis at the first attendance, but a decision regarding management should be made. The problem is an individual one in that treatment is being advised for one particular child, and not for a disease; it might be added that the attitude of the parents to the child's disability must be considered. The essential point is to select the children who will benefit from appendicectomy. No great harm results if operation is advised for mesenteric adenitis under a mistaken diagnosis of appendicitis. As will be seen from the follow-up, appendicectomy almost always relieves the symptoms. If, on the other hand, the child has periodic disease, the symptoms tend to persist after appendicectomy. Even so, operation may have to be advised as the only way of positively excluding chronic appendicitis.

Assuming that a diagnosis of mesenteric adenitis has been made, my present practice is to treat these children conservatively in the first instance. After a short period of treatment and observation, operation is advised without delay if there is no improvement. Operation is particularly advised in two circumstances: first, when the pain is causing appreciable disability or is increasing in severity; secondly, in patients who have been admitted as an urgency it is often better to perform appendicectomy whilst the child is still in hospital.

The conservative treatment consists of a search for any causes of stress, reassurance, and an explanation of the condition to the parents. A low fat diet is sometimes advised (Farquhar, 1956). The best drug for controlling the episodes of pain and pyrexia is aspirin (Kempton, 1956). Antihistamines often relieve the pain even when there is no obvious allergic basis for the symptoms. Farrer (1956) advised a high-protein diet and belladonna for periodic disease.

As a result of this survey, it appears that operation should not be postponed indefinitely if the patient does not respond to medical treatment. The attacks of pain may cause considerable anxiety to the parents, as it is difficult to exclude chronic appendicitis with certainty. Most patients are cured after appendicectomy, the response is usually dramatic, pain is relieved, and the general condition improves.

PROGNOSIS

The results of a long-term follow-up are seen in *Table II*. All the patients were followed up from 3 to 6 years after discharge, apart from one boy in the operative group who could not be traced.

Table II.—FOLLOW-UP OF 48 PATIENTS WITH MESENTERIC ADENITIS AFTER 3–6 YEARS

Symptoms	*Operative Cases*	*Treated Medically*
No symptoms	20	14
Mild abdominal pain	1	5
Some pain, not like previous pain	3	—
No different	1	2
No pain, but off colour	1	1

Most of the children who had an appendicectomy were well and symptom-free. Only one was not improved. His history, on review, is more suggestive of the periodic syndrome than mesenteric adenitis. One child was off colour but had no pain, and another had only very slight pain. Of the three children whose pain had changed, two have migraine as previously noted and the other has had a history of periodic pain since infancy.

In the conservatively treated group the two patients whose symptoms are unchanged have, again in retrospect, a history of the periodic syndrome. Six children still have occasional attacks of mild pain, usually not enough to interfere with their normal activities, and they show a general tendency to improve. Two have since had an appendicectomy, but unfortunately details are not available; both are well now. One girl who has no abdominal pain now has a history of migraine.

Two aspects of the prognosis require consideration. First is the question of recurrence after operation. Most authors, summarized by Ward-McQuaid (1951), found a recurrence rate of 20–25 per cent. In this series 16 per cent have some symptoms and 12 per cent have abdominal pain; this is after cases of migraine and the periodic syndrome have been excluded. Secondly, is the question of whether appendicectomy has any influence on the course of the disease. Aird (1945) states that it is self-limiting, and this series supports his view. Although approximately one-quarter of the patients still have some pain, they are considerably improved, and the pain is not sufficient to justify operation. Almost three-quarters are free from pain. The results after appendicectomy are rather better, and, in view of the striking improvement after operation, there seems to be good reason for advising it in children who are not responding satisfactorily to medical treatment.

SUMMARY AND CONCLUSIONS

Forty-nine children who had a diagnosis of mesenteric adenitis made between three and six years ago are reviewed. Twenty-seven patients had the diagnosis proved by operation.

The appearances at operation are described and the aetiology is discussed. The cause of non-specific mesenteric adenitis is not known, but it is possibly a virus infection.

In most cases the disease occurs in a chronic form. Before making a diagnosis of mesenteric adenitis, migraine should be excluded. The relationship between mesenteric adenitis and the periodic syndrome is discussed. Although the two diseases are distinct, there may be some overlap.

Each case must be treated individually on its own merits. The indications for appendicectomy are discussed; after operation the symptoms are relieved, and there is no evidence that the pain recurs after appendicectomy.

In the medically treated cases the symptoms settle slowly, and most children become free from pain after a few months.

A follow-up is presented; most of the patients are well, but the results are slightly better in the patients who have had an appendicectomy.

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REFERENCES

AIRD, I. (1945), *Brit. med. J.*, **2**, 680.
ASENCIO, F. (1949), *J. int. Coll. Surg.*, **12**, 702.
BRENNEMANN, J. (1921), *Amer. J. Dis. Child.*, **22**, 493.
British Medical Journal (Leader) (1957), **2**, 398.
COLLINS, D. C. (1955), *Miss. V. med. J.*, **77**, 146.
DONHAUSER, J. L. (1957), *Arch. Surg., Chicago*, **74**, 528.
FARQUHAR, M. G. (1956), *Brit. med. J.*, **1**, 1082.
FARRER, J. A. (1956), *Ibid.*, **1**, 294.
FITZGERALD, M. J. T. (1956), *Irish J. med. Sci.*, **365**, 205.
FITZSIMONS, J. (1946), *N.Z. med. J.*, **45**, 248.
FOSTER, A. K. (1938), *Arch. Surg., Chicago*, **36**, 28.
— — (1939), *Ibid.*, **38**, 131.
FREEMAN, L. (1923), *Surg. Gynec. Obstet.*, **37**, 149.
— — (1929), *Ann. Surg.*, **90**, 618.
GAGE, I. M. (1939), *Ibid.*, **109**, 827.
GOLDBERG, S. L., and NATHANSON, I. T. (1934), *Amer. J. Surg.*, **25**, 35.
GROSS, R. E. (1953), *Surgery of Infancy and Childhood*, 261. Philadelphia: Saunders.
HAMILTON, C. K. J. (1951), *Proc. R. Soc. Med.*, **44**, 719.
HEUSSER, H. (1923), *Beitr. klin. Chir.*, **130**, 85.
IRELAND, J. (1938), *Arch. Surg., Chicago*, **36**, 292.
KEMPTON, J. J. (1956), *Brit. med. J.*, **1**, 83.
KLEIN, W. (1938), *Arch. Surg., Chicago*, **36**, 571.
MADIGAN, H. S., and COFFEY, R. J. (1950), *Ibid.*, **60**, 1122.
POSTELTHWAIT, R. W., and CAMPBELL, F. H. (1949), *Ibid.*, **59**, 92.
RAFTERY, A., TRAFAS, P. C., and MCCLURE, R. D. (1950), *Ann. Surg.*, **132**, 720.
REIMANN, H. A. (1957), *New Engl. J. Med.*, **256**, 652.
ROSENBURG, S. (1937), *Arch. Surg., Chicago*, **35**, 1031.
STRANG, R. (1957), *Scot. med. J.*, **2**, 425.
STRUTHERS, J. W. (1921), *Edinb. med. J.*, **27**, 22.
WARD-MCQUAID, J. N. (1951), *Lancet*, **2**, 524.
WEBSTER, R. E., and MADORE, P. (1949), *Gastroenterology*, **13**, 160.
WHITE, C. S., and COLLINS, J. L. (1936), *J. Amer. med. Ass.*, **107**, 1023.
WILENSKY, A. O. (1920), *Med. Rec.*, **98**, 220.
— — and HAHN, L. J. (1926), *Ann. Surg.*, **83**, 812.

RETRO-ANASTOMOTIC HERNIA

EIGHT CASES OF INTERNAL HERNIA FOLLOWING GASTROJEJUNAL ANASTOMOSIS, WITH A REVIEW OF THE LITERATURE

By J. R. G. BASTABLE* and P. E. HUDDY†

Following antecolic or retrocolic gastrojejunal anastomosis, an artificial ring is made behind the stoma and the anastomosing loop of the jejunum. Through this space small intestine or omentum may pass and undergo the complications of incarceration or strangulation. The condition is frequently called retro-anastomotic hernia, and is sometimes named 'Petersen's hernia' in German and American articles.

HISTORICAL REVIEW

The first case was reported by Buddee (1897), where most of the small intestine had passed through the hiatus following a retrocolic gastro-enterostomy. Petersen (1900) gave the first full account of the condition, and described 2 cases where most of the small intestine had passed through the gap after antecolic partial gastrectomy and after posterior gastro-enterostomy. The first cases of afferent loop herniation were described by Kelling (1900) after retrocolic gastro-enterostomy, and by Weber (1901) after antecolic gastrojejunostomy. Fromme (1920) found in the literature 4 cases following antecolic, and 18 after retrocolic gastrojejunal anastomosis. Himmelmann (1933) in a review collected 8 cases after antecolic and 36 after retrocolic anastomosis, of which the majority followed gastro-enterostomy. After antecolic anastomosis, 6 were of efferent loop type, 2 involved the afferent loop, and in 1 the loop and direction were unstated (1 case—Weber, 1901—had a double type with the afferent loop passing in one direction and the efferent loop in the other direction). In the cases following retrocolic anastomosis, the afferent loop was involved only in 1 case, and in at least 2 cases small intestine had passed through the hiatus in a left-to-right direction. Stammers (1952, 1954) described 12 cases, of which all but one followed antecolic partial gastrectomy.

Table I.—Incidence of Types of Hernia

	Anastomosis	Direction of Bowel	Number of Cases
Afferent loop	Antecolic	Right to left	34
	Retrocolic	R. to L.	3
	Unstated	R. to L.	1
	Unstated	Unstated	3
			41
Efferent type	Antecolic	R. to L.	17
	Antecolic	L. to R.	5*
	Antecolic	Unstated	1
	Retrocolic	R. to L.	31
	Retrocolic	L. to R.	3
	Retrocolic	Unstated	9
	Unstated	L. to R.	3
	Unstated	Unstated	3
			72
Omentum	Antecolic	R. to L.	2*
		Total patients	114

* In one case both small intestine and omentum had herniated.

We are presenting 8 cases, and will discuss them together with 106 other cases recorded since the report of Himmelmann (1933) or not included in his article. Since no review of published cases has been made since 1933, we feel that this larger series may reveal an incidence rather higher than generally supposed, and that an analysis of these cases may be of practical value.

Table I shows the incidence of the different types of herniation. The 'efferent type' has included all cases where bowel distal to the stoma has herniated.

CASE REPORTS

Case 1.—A. C., a male aged 49 years, had an antecolic partial gastrectomy performed for a chronic duodenal ulcer on June 7, 1949; the proximal loop was sutured to the lesser curve and the anastomosis was made with a Hofmeister valve. Post-operatively his course was uneventful and he was discharged home symptom-free.

History.—At 6 p.m. on the fifteenth post-operative day he had a sudden onset of severe colicky abdominal pain, felt mainly in the epigastrium. On three occasions he vomited small quantities of colourless fluid.

On Examination.—At examination on admission at 11 p.m. on that day he was shouting because of severe pain. His general condition was fair. A firm, tender, non-resonant swelling was felt under the left costal margin. The lump moved on respiration, but the upper border could not be palpated, and it was described as feeling "like a spleen".

By 1.30 a.m. next day his pain was still agonizing. His pulse was 100 per minute and blood-pressure 115/70. The mass was not now felt owing to generalized board-like rigidity. A diagnosis of general peritonitis was made.

At Operation.—At 3 a.m. the abdomen was opened through a midline incision. On opening the peritoneum, gas and copious bile-stained fluid escaped. The afferent loop was found to have slipped from right to left (through the retro-anastomotic hiatus) behind the axis of the mesentery of the anastomotic loop, which was constricting it. There was a perforation 5 cm. in diameter in the proximal loop at 7 cm. from the duodenojejunal flexure. The stoma was intact, but the afferent loop had separated from its attachment at the lesser curve. The efferent loop was not obstructed, and the duodenal stump was intact. The rent in the jejunum was sutured, the hernia reduced, and the afferent loop sutured in the reduced position. After peritoneal toilet, the incision was closed.

Post-operatively the patient's general condition was initially satisfactory, but after a few hours he passed into a state of shock, which failed to respond to plasma and blood transfusions. He died at 5.30 a.m. on June 24 (26½ hours after operation).

Case 2.—P. B., a male aged 54 years, had a chronic prepyloric ulcer adherent to the pancreas. On March 5, 1952, he had a two-thirds partial gastrectomy with an antecolic gastrojejunal anastomosis made with a Hofmeister valve. Convalescence was uneventful.

He was re-admitted on Oct. 25. He was confused and a clear history was difficult to obtain. Six days before he had had vague abdominal pain, but continued at work

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until four days before admission. Three days before admission he awakened early with severe colicky abdominal pain, which persisted as a less severe pain. He vomited small quantities. The bowels had not opened for a week.

On Examination.—He was confused and extremely ill. His temperature was 97° F. and his pulse-rate 100. He was cyanosed and in slow auricular fibrillation, with a blood-pressure of 70/40, and respiratory rate of 50 per min. A large number of moist sounds were heard at the lung bases. The abdomen was moderately distended, with generalized rigidity and tenderness, most marked in the right iliac fossa. No bowel-sounds were heard. A diagnosis of peritonitis due to appendicitis was made.

He was treated with intravenous dextran and gastric aspiration, which produced 700 ml. of fluid, coloured like 'anchovy sauce'. After 1500 ml. of dextran his blood-pressure rose to 90/60, he was a better colour, and more rational. The aspirate had by then become heavily blood-stained. He was prepared for operation, but died during the induction of anæsthesia.

At post-mortem examination there was generalized seropurulent peritonitis. The efferent loop of jejunum had passed through the retro-anastomotic hiatus and become strangulated. The direction was not stated.

Case 3.—E. D., a male aged 56 years, had a chronic duodenal ulcer. On Oct. 4, 1955 partial gastrectomy was performed with an isoperistaltic, antecolic, gastrojejunal anastomosis. Convalescence was uneventful.

When admitted 22 months later on Aug. 16, 1957, he gave a history of having had mild recurrent attacks of abdominal pain, occurring since operation. There had been no vomiting. Forty-eight hours before admission he had had a sudden onset of an agonizing lower abdominal pain, which got worse at times. The pain was mainly right-sided and later radiated to the back. He had repeated retching, and vomited small quantities of whitish frothy fluid on ten occasions. During this period he passed flatus on two occasions, but had no bowel action.

On Examination.—He was sweating, and in fair general condition. The temperature was 97° F., pulse 56 per minute, and the blood-pressure 135/80. His tongue was dry and furred. There were visible peristalsis and tenderness and rigidity in the upper abdomen. A diagnosis of perforation of a stomal ulcer was made.

At Operation (Aug. 16).—After induction of anæsthesia, a swelling was seen and felt under the upper part of the left rectus abdominis muscle. The lump was oval and 4 in. long in the greater axis. A right upper paramedian incision was made. There was no free fluid. There were adhesions between the stomach remnant and its surroundings. The afferent and efferent loops of jejunum were normal. About half the small intestine had passed from right to left through the retro-anastomotic hiatus, and one loop of the herniated bowel had undergone torsion. A second small loop had also herniated through the hiatus. The herniated gut was congested and purple in colour. Reduction was easy and the congestion rapidly went. The upper part of the root of the mesentery was œdematous. The afferent and efferent jejunal loops were sutured to the mesocolon. The post-operative course was uneventful and he was well when seen in the follow-through clinic three months later.

Case 4.—D. G., a male aged 37 years, had a partial gastrectomy performed for a chronic duodenal ulcer on July 12, 1955. The gastrojejunal anastomosis was antecolic, with a Hofmeister valve.

Convalescence was uninterrupted until the sixth post-operative day when he complained of lower abdominal pain, which was constant and severe. He vomited three times.

On Examination the following day he had tenderness and rigidity in the right iliac fossa. His pulse had risen from 88 to 130 and the blood-pressure was 130/40. A diagnosis of a ruptured duodenal stump was made.

At Operation (July 19).—There was a large quantity of turbid bile-stained free fluid in the peritoneal cavity. The duodenal stump was leaking and the afferent loop was obstructed, having passed from right to left behind the efferent loop. It was neither perforated nor gangrenous. The afferent loop was reduced, and a side-to-side anastomosis made between afferent and efferent loops. The duodenal stump was oversewn with omentum and the abdomen closed with drainage. His convalescence was slow owing to an infected atelectasis of the right lower lobe. He was discharged on Aug. 23 and was well when seen three months later.

Case 5.—R. B., a male aged 58 years, had a two-thirds partial gastrectomy performed on April 8, 1957, for a chronic duodenal ulcer. An antecolic gastrojejunal anastomosis was made with the afferent loop to the lesser curve. Post-operatively he developed an infected atelectasis of the left lower lobe. Following breakfast, on the eleventh post-operative day, he complained of epigastric fullness, followed by gradually increasing, constant, central abdominal pain, made worse by movement. At midday he vomited food, and this was followed by six fluid vomits, the last two containing bile. The bowels were opened before the onset of pain, but neither fæces nor flatus was passed afterwards.

On Examination.—The pulse was 92 per minute, and there was distension and tenderness in the left upper abdomen. No bowel-sounds were heard. He was given 30 ml. water by mouth hourly and gastric aspiration produced between 7 ml. and 110 ml. of yellow, fæculent fluid. On the following day his pain was less severe, temperature was 97° F., and pulse 82. He looked ill and his tongue was dry and furred. There was slight generalized abdominal tenderness, which was more marked in the left upper abdomen. There was slight rigidity of the left upper rectus, and a slight fullness was to be seen at this site, where an indefinite lump could be felt. The abdomen was slightly distended and bowel-sounds were reduced in frequency. The gastric aspirate was greater than the intake. Diagnosis of intestinal obstruction was made, and it was considered that it might have been due to a retro-anastomotic hernia.

At Operation (April 20).—A left upper paramedian incision was made. There was a small quantity of yellow, turbid, intraperitoneal fluid. The stomach, stoma, and efferent loop were normal. The dilated and congested afferent loop lay behind the efferent loop, having passed from right to left through the retro-anastomotic ring. Entero-enterostomy was performed between the afferent and efferent loops. The incision was closed with drainage. The post-operative course was uneventful and he was discharged symptomless.

Case 6.—D. W., a male aged 35 years, had a valved antecolic partial gastrectomy performed on Nov. 14, 1951, for a chronic, lesser-curve gastric ulcer. The afferent loop was long. Post-operatively he had a lobular atelectasis of the lower lobe of the right lung which responded to treatment. He was discharged symptomless.

At 8 p.m. on Dec. 11 (27 days after operation) following a large meal of tinned herrings he experienced the sudden onset of severe, continuous abdominal pain. He vomited two or three times without relief of pain. His bowels were not open and he passed no flatus. He was admitted at 3 a.m. on Dec. 12.

On Examination.—He was rolling in agony, and was retching and hiccupping. He was sweating, and was pale and grey, with a subnormal temperature and a pulse-rate of 60. The tongue was dry and furred. The abdomen was generally distended, with epigastric tenderness and rigidity. No abnormality was felt rectally. A provisional diagnosis of intestinal colic, possibly due to dietary indiscretion, was made, and he was treated by gastric aspiration and intravenous fluids. At 8 a.m. the pain was more severe and his pulse-rate had risen to 116. There was generalized

abdominal tenderness maximal in the right iliac fossa, with rigidity in the epigastrium. Some discoloration was noted around the umbilicus. No bowel-sounds were heard. No blood or bile was present in the gastric aspirate. A radiograph of the abdomen showed no free gas or fluid levels. A diagnosis of afferent loop obstruction was made.

OPERATION was undertaken at 1 p.m. on Dec. 12. The peritoneum contained a large quantity of pale yellow fluid tinged with green. The efferent loop was normal and on its right the afferent loop was found to be dark purple in colour, and herniated from right to left through the retro-anastomotic hiatus. The afferent loop was collapsed and flabby and had undergone torsion. No perforation was found, but at one area the seromuscular coat was ruptured. The afferent loop was reduced by drawing it to the right behind the efferent loop. The duodenum was not dilated, its stump was intact. Convalescence was uneventful.

Case 7.—W. W., a male aged 44 years, had a partial gastrectomy performed on Sept. 7, 1956, for a chronic ulcer on the posterior wall of the first part of the duodenum. Following operation, he was at first well and able to eat a nearly full-size meal without symptoms. Four months after operation he had an attack of influenza and following this he began to vomit bile. Vomiting was in bouts lasting one to three days, with periods of freedom of one to two weeks. During the vomiting attack he experienced nausea one and a half hours after meals, and vomited about thirty minutes later, while his motions were soft and like putty in consistency and colour. He lost 2½ st. in weight. His appetite became poor and he could eat only half a normal size meal. No abnormality was found on repeated examination. Barium meal and follow-through showed an irregularity at the stoma, which was considered to be suggestive of an ulcer crater; gastric emptying was rapid and passage through the small intestine was normal. Hæmoglobin 15·8 g. per cent. Stools contained undigested meat fibres and no excess of fat. The symptoms persisted without change. Diagnosis was gastrojejunal ulceration.

OPERATION was performed on Nov. 21, 1958.—The previous right upper paramedian incision was reopened. Several dense adhesions were divided. At least half the stomach remained, with an antecolic gastrojejunal anastomosis, using the full width of the stomach and a short Hofmeister valve on the lesser-curve end of the stoma. The afferent jejunal loop had been united to the lesser curvature, and the stoma was rotated so that the greater curvature lay anteriorly. The afferent jejunal loop was 9 in. long, had passed from right to left through the retro-anastomotic hiatus, and was kinked at its attachment to the stomach. Both the afferent loop and the duodenum were dilated. The stoma was divided and the jejunum closed. Half the gastric remnant was excised, and a retrocolic, no-loop gastrojejunal anastomosis made. The post-operative course was uneventful. Four weeks later he was able to eat a nearly full-size meal, and had had no further vomiting.

Case 8.—M. G., a male aged 45 years, had a persistent melæna from a chronic ulcer of the upper border of the first part of the duodenum, intimately adherent to the hepatic artery and common bile-duct. On Oct. 25, 1958 partial gastrectomy was performed with a retrocolic, short-loop gastrojejunal anastomosis, the afferent loop being anastomosed to the lesser curvature.

Post-operatively he developed atelectasis of the left lower lobe. On the fifteenth post-operative day he started to vomit bile after meals, and from the thirty-third to the forty-third days small quantities of bile discharged from the centre of the incision. He was discharged on Dec. 8. After discharge bile vomiting of one or two cupfuls after each meal continued for one day each week. He was able to eat a half-sized meal before getting a feeling of distension in the upper abdomen. These symptoms continued until on Jan. 5, 1959, he began to vomit all ingested food mixed with bile.

ON EXAMINATION.—He was dehydrated. A slight discharge of pus came from the centre of the incision, and he was tender in the right upper abdomen. Gastric suction produced large amounts of bile and ingested water did not leave the stomach. On Jan. 8 operation was performed. The previous incision was reopened and a collection of subcutaneous pus led to an abscess cavity in the vicinity of the duodenal stump. The stoma was found to be rotated with the greater curvature lying anteriorly. The afferent jejunal loop had passed from right to left through the retro-anastomotic hiatus and was considerably dilated. An entero-enterostomy was made between the afferent and efferent loops, and the abscess drained. The post-operative course was uneventful.

DISCUSSION

The Retro-anastomotic Hiatus.—The boundaries of the retro-anastomotic hiatus after retrocolic gastrojejunostomy are superiorly the transverse mesocolon, posteriorly the peritoneum of the posterior abdominal wall and ligament of Treitz, inferiorly the afferent jejunal loop and its mesentery, and anteriorly the afferent jejunal loop and its mesentery, together with the stoma. After antecolic gastrojejunostomy the boundaries are the same, except that superiorly and anteriorly there are the entire length of the transverse mesocolon, the transverse colon, and the greater omentum; a second space is also formed above the transverse mesocolon and colon, through which herniation might occur.

The size of the retro-anastomotic hiatus after retrocolic gastrojejunal anastomosis is normally small, but depends on a number of different factors. A long afferent jejunal limb makes a J loop and forms the greater part of a large ring, while a short afferent loop produces a small slit-like aperture with a firmer anterior wall. A short mesentery will be relatively taut and form the anterior margin of the ring. In a small number of patients the duodeno-jejunal flexure lies lower than normal, resulting in a vertically elongated hiatus. The length of the superior wall depends on the distance of the mesocolic opening from the root of the mesocolon. Following an extensive partial gastrectomy, the mesocolon may be pulled up as a cone, enlarging the hiatus in a vertical direction.

Antecolic anastomosis produces a large opening, whose exact size depends on the length of the afferent loop and its mesentery, the bulk of the remaining greater omentum, and the size of the transverse colon. The dimensions of the aperture in either type of anastomosis may be modified by the presence of adhesions.

Incidence.—The incidence of retro-anastomotic herniation has been difficult to assess, because few reports mentioned the number of operations performed. Afferent loop herniation after antecolic partial gastrectomy was reported as occurring in 2 of 98 operations by Hsü, Hu, and Hsü (1956), in 3 of 130 by Mimpriss and Birt (1948), and in 2 of 220 in our series. When the type of anastomosis was not specified, Stammers (1952) found 1 case of afferent loop herniation in over 600 operations, and Warren (1954) 2 in 120. Herniation of efferent type after partial gastrectomy occurred in 1 of 120 cases of Warren (1954), 1 in 800 of Lefèvre (1947), and 2 in 420 in our cases. Viannay (1938) recorded 1 hernia of efferent type occurring in just over 100 gastro-enterostomy operations of unspecified type.

These figures have failed to take account of many large series of operations where this complication has not occurred. On the other hand, cases might not have been reported, because single cases were not thought worthy of publication or the condition had not been discovered, as routine post-mortem examinations were not done and the death was attributed to duodenal stump rupture or intestinal obstruction from other causes.

Aetiology.—Several reasons have been suggested to account for the occurrence of the herniation and for the preponderance of cases in which the herniated bowel has passed from right to left through the retro-anastomotic hiatus. The theories are:—

1. Removal of gauze placed behind the anastomosing loop, carrying bowel through the hiatus (Petersen, 1900).

2. Small-intestine contractions making the bowel narrow and rigid, and more likely to insinuate itself into openings (Reischauer).

3. Changes of intra-abdominal pressure, produced by vomiting, coughing, etc., forcing small intestine through the hiatus.

4. Paralysis of the left side of the diaphragm after abdominal operations, causing the intestines to be pushed up and to the left (Lochard).

5. That the loop attached to the lesser curvature of the stomach remnant would be relatively fixed, and that the loop attached to the greater curvature of the stomach would be pushed towards the midline by gastric peristalsis and next slide back to its normal position, when it might enter the retro-anastomotic hiatus (Roux, Nègre, and Marchal, 1954).

6. After antecolic anastomosis, the peristalsis of the transverse colon carrying a loop of small intestine through the hiatus from right to left (Bortuzzo, 1955).

Most of these theories would seem unsatisfactory, but the length of the afferent loop and its anatomical relations to stomach and colon would appear to influence the type and direction of the hernia.

The proximal loop was long in nearly five-sixths of the cases of afferent loop herniation, suggesting that a redundant loop would pass through the retro-anastomotic hiatus, while a short loop would be taut and unable to herniate. In efferent loop herniation the length of the afferent loop was found to be either long or short in an equal number of cases. Only 2 cases of afferent loop herniation have been reported, where the proximal loop had been united to the greater curvature in making the anastomosis. On the other hand six-sevenths of the cases of afferent loop herniation followed antecolic partial gastrectomy with anastomosis of the proximal loop to the lesser curvature. After partial gastrectomy the direction of the herniated gut was found to be from right to left in all cases involving the afferent loop, and in four-fifths of cases of the efferent type.

Pathology.—In the reported cases of efferent type the herniation has usually started in the jejunum just distal to the *stoma*, while in a smaller number the herniation has started with the *efferent jejunal loop* or the ileum. In 33 cases most of the small intestine had passed through the hiatus, except the last 1–2 ft. of ileum, which had been prevented from herniating by the attachment of the cæcum to the posterior abdominal wall. In several the terminal ileum had been stretched as a taut band between the hiatus and the cæcum. In 2 further cases (Borsato, 1949) the cæcum and ascending colon had a mesentery, and the herniated bowel included the cæcum in one case and in the other the cæcum and part of the ascending colon.

As with all hernias, the usual complications have occurred, culminating in gangrene and perforation. At the time of operation the herniated intestine was more frequently gangrenous or perforated in afferent loop herniation—in 37 fully described cases of this type 17 showed one or other of these changes, including 2 cases where the duodenal stump had ruptured, while in only 3 cases was the herniated bowel of normal appearance. In the efferent type the herniated bowel was gangrenous or perforated in only 8 of 63 fully described cases, while in 10 cases it appeared normal. In 12 cases of efferent type of herniation changes were found in the afferent loop, caused by pressure or obstruction, amounting to distension in 4, strangulation in 2, gangrene or perforation in 4, and rupture of the duodenal stump in 2 cases. In all types of hernia rupture of the duodenal stump was especially found in patients in the early post-operative period.

The strangulating agent was stated in only a few cases and in those described was the afferent loop in herniation of the efferent type, and the mesentery of the anastomosing loop in afferent loop herniation. Three further factors have played a part; as in either variety a closed loop could be formed, in the efferent type hernia torsion of the mesentery could obstruct the mesenteric vessels, and in cases of afferent loop herniation there could be kinking at the stoma. The size of the ring would be expected to influence the onset of gangrene, but in the collected series a short proximal loop did not appear to produce a higher instance of gangrene in the efferent type of hernia. On the other hand, a short proximal loop after antecolic partial gastrectomy was associated with gangrene of the herniated afferent loop in 3 out of 4 cases, while gangrene occurred in 9 out of 21 cases with a long loop. After antecolic anastomosis with the efferent type of herniation there was no case which went on to the stage of gangrene.

Other pathological changes were found to be associated with these hernias. In 1 case in every 10 cases the stoma and a variable part of the stomach had undergone torsion, and there was the same incidence of volvulus of the herniated loop in either type of herniation. In 4 cases of efferent type of herniation the superior mesenteric vein was found to be obstructed, while in 1 of these cases the accompanying artery was also obstructed, and in this case, although the circulation returned after reduction, both vessels later thrombosed. Distension of the afferent loop and duodenum may result in back-pressure on the bile and pancreatic ducts. The serum-amylase was estimated in 2 patients with herniation of the afferent loop and in both cases it was elevated. In 1 case in every 12 cases there was either jaundice or a dilatation of the common bile-duct or gall-bladder.

Clinical Features.—The age and sex incidence did not appear to be aetiological factors since they did not differ from those in published series of gastric operations. Similarly the initial disease had no apparent bearing on the occurrence of the hernia.

The type of operation had an influence on the type of hernia. In the cases reported since Himmelmann (1933) gastro-enterostomy was followed only by the efferent type of herniation, and the vast majority followed the retrocolic operation, presumably because this was by far the commonest method employed. After retrocolic partial gastrectomy most of the hernias were of efferent type. Nearly all cases of afferent loop herniation followed antecolic partial gastrectomy, but one-third of the hernias after this operation were of the efferent type.

The time from the initial operation to the onset of symptoms due to the herniation varied from 1 day to 13 years. About half of the cases occurred within the first 28 days of operation, a quarter between 1 month and 12 months, and a quarter over 1 year. Of the cases occurring in the first month three-quarters were during the first fortnight. There was a slightly earlier incidence after antecolic anastomosis.

The clinical features differed in the afferent and efferent types of hernia, and have been summarized in *Table II*.

Table II.—Clinical Features of Types of Hernia

	Afferent Loop	Efferent Type
Pain	Epigastric Constant	Generalized abdominal Colicky
Vomiting or gastric aspirate	Not marked Small quantities Bile absent Blood absent	Many Large quantities Bile absent in one-third cases Blood rarely present
Tenderness	In left upper abdomen	In two-thirds cases usually generalized
Distension	None before peritonitis	Generalized in half cases
Rigidity	Absent before peritonitis	Absent before peritonitis
A lump	In left upper abdomen in one-third cases	Rarely present in the left upper abdomen
Plain radiographs	No fluid levels. Soft tissue mass in left upper abdomen in ¼ cases	Fluid levels present

Afferent Loop Hernias.—The presenting symptom was almost always pain, which was of sudden onset in the majority of the patients. The site of the pain was most often in the upper abdomen, usually in the epigastrium, but sometimes in the left upper quadrant, or at the level of the umbilicus, while in 2 patients the pain was generalized. The pain was severe in the majority of the patients, but in 6 patients the pain was initially slight, becoming severe later in 4 of these. In 2 patients the pain was eased by gastric suction. The pain was constant in most cases, but in 8 patients was initially colicky in character, and in 2 an initial colicky pain later became constant. In 2 patients the pain remitted for a period of up to 12 hours, and a further 2 patients were screaming or writhing with pain. In 2 patients the pain radiated into the back.

Vomiting occurred in almost all cases of afferent loop herniation, varying between one and six times, and usually occurring within a few hours of the onset of symptoms. The quantities were sometimes large, and in only one-tenth of cases was bile present in the vomit or gastric aspirate, while in no case was there frank blood.

Half the patients were constipated, and 1 patient had diarrhœa as the first symptom. Belching, hiccups, and retching were infrequent.

The general condition on admission varied widely from looking well to being moribund. The majority were apyrexial, but in a few the temperature was subnormal, and these patients were usually seen early after an acute onset. The pulse was usually raised and tended to become more rapid during a period of observation; a few patients admitted in shock had a low blood-pressure. Four patients were jaundiced.

Abdominal tenderness was present in all cases, and before the onset of peritonitis was found in the upper abdomen, usually to the left of the midline. Distension was absent prior to the onset of peritonitis. Rigidity was an important physical sign and indicated the presence of peritonitis.

A lump was felt in rather more than one-third of the cases, and was present in the left upper abdomen. The swelling was usually described as mobile, elastic, and about the size of a fist. In several cases a swelling was seen on inspection, and in 2 patients could no longer be seen after the onset of rigidity.

Efferent Type Hernias.—The presenting symptom was pain in only two-thirds of the cases, and in the remainder nausea and vomiting, which was usually followed by pain after a variable interval. The onset of the pain was usually sudden in patients with retrocolic anastomosis, but an equal number had sudden and gradual onsets when there was an antecolic anastomosis. The site of the pain was usually epigastric or across the centre of the abdomen, while a few patients had pain in some part of the lower abdomen, and 4 had pain in the back on lying flat in bed. The pain was generally severe, especially when the hernia followed retrocolic anastomosis. The character of the pain was colicky in two-thirds of the cases, and either constant or constant and colicky in the remainder of the patients. Four patients were screaming or writhing with pain, and in only 1 case relief of the pain followed gastric suction. Vomiting occurred in seven-eighths of the patients, usually several times, and usually in large amounts. Vomiting was the presenting symptom in one-third of the patients, and in the remainder usually occurred within a few hours of the onset of symptoms. The vomit contained bile in two-thirds of the cases, while in the remainder its absence was explained by obstruction of the afferent loop. Blood was present in the vomit in 7 cases. Belching, hiccups, and retching were infrequent.

Several patients had a bowel action after the onset of the pain. One patient had bloody diarrhœa as the first symptom, another developed diarrhœa soon after the onset of abdominal pain, and a third patient had bloody diarrhœa several hours after the onset of pain.

The general condition of the patients on admission was similar to those with afferent loop herniations. Two patients had tetany, and both had had incessant vomiting. Four patients were jaundiced.

Abdominal tenderness was found in two-thirds of the patients, and was usually generalized, but in a

lesser number was in the upper abdomen. Distension was present in half the cases, and was usually generalized, but occasionally in the upper abdomen. Rigidity was only found in patients with peritonitis. An intra-abdominal lump was a comparative rarity, occurring in 5 patients in the left upper abdomen, and in 1 case at the umbilicus. In a few patients bowel-sounds were increased, and at operation only slight congestion of the herniated bowel was found usually.

Chronic or Recurrent Cases.—In contradistinction to the typical case, 1 patient in every 8 patients had a different clinical course. Six cases with efferent loop hernia were in this category, of which 1 case (Hsü and others, 1956) had two preceding attacks of moderately severe pain and distension, each with spontaneous relief. The remainder had mild symptoms, which commenced between five days and four months after operation, and lasted between sixteen days and ten months before further operation. Three patients had recurrent bile vomiting after meals, and of these only 1 patient subsequently developed complete obstruction. One patient (Reiner, 1955) had fullness and nausea after meals, while 1 patient had recurrent slight pain and attacks of vomiting (Omez, Corbeau, and Coornaert, 1950). Of the 8 cases with efferent loop herniation, one had no symptoms recorded, 5 had recurrent attacks of abdominal pain with nausea or vomiting, and 2 had repeated painless vomits. The onset of symptoms varied between four days and six months from operation, and the period until re-operation was between three weeks and three and a half years.

In both types of herniation, tenderness and slight distension were sometimes found on examination, but no lump was found. There was no correlation with either an antecolic or a retrocolic anastomosis.

Investigations.—Straight radiographs of the abdomen showed fluid levels in the majority of the efferent type of hernias. In contrast, in afferent loop herniation a soft-tissue mass was seen in a quarter of the cases, while the only case to show multiple fluid levels had general peritonitis.

Barium meals were performed in 8 cases of afferent loop herniation. Abnormalities found were: in 2 negligible gastric emptying, in 2 partial torsion of gastric remnant, in 1 the efferent loop lying anteriorly, in 1 a mass below the stoma, in 1 no definite abnormality, and in 1 stomal irregularity suggestive of an ulcer crater. Seven opaque meal examinations were done in the efferent type of hernia, and in 3 the stomach emptied normally; in the remaining cases the stomach failed to empty or slowly emptied, in 1 case the jejunum was seen to be obstructed 5 in. below the stoma, and in another there was a soft-tissue mass adjacent to the efferent loop.

Pre-operative Diagnosis.—A variety of pre-operative diagnoses were made and only occasionally was retro-anastomotic hernia considered, usually when the condition had been previously experienced. In cases of afferent loop herniation with perforation, the diagnosis was usually general peritonitis secondary to perforated stomal ulcer, or dehiscence of the stoma or duodenal stump. In the efferent loop herniations the diagnosis was intestinal obstruction in about half the cases, and in the remainder a variety of other acute abdominal conditions.

Operative Findings and Treatment.—In both afferent and efferent loop herniations the large majority of patients had free intraperitoneal fluid, which was occasionally blood-stained, and which contained bile in the cases with a perforation of the afferent loop or a rupture of the duodenal stump.

Difficulty was experienced in several cases of afferent loop herniation in finding the strangulated loop, and in some cases the rotation of the stoma was a guide to the site of the obstruction. In the efferent type the herniated bowel frequently was found to be lying on the left side of the peritoneal cavity.

Herniations of the afferent loop were reduced and it was sometimes necessary to untwist a volvulus or to aspirate the loop before reduction was possible. In about one-third of cases fixation was achieved by an entero-anastomosis between efferent and afferent loops. Perforation was treated by suture, and gangrene by resection. The efferent type of hernias were also treated by reduction and it was frequently necessary to untwist the volvulus of the herniated bowel before this was possible.

In some cases the retro-anastomotic ring was closed, usually by suturing the afferent loop or mesentery to the transverse mesocolon and posterior parietal peritoneum.

Mortality.—The mortality-rate was high for both types of herniation, being 36 per cent for the afferent and 40 per cent for the efferent variety.

There appeared to be no correlation between the mortality-rate and the sex or age of the patient. The mortality was higher in those hernias occurring while the patient was still in hospital after the initial operation, due in part to the failure to treat the condition by a second operation. The other factor of importance in the mortality was delay in operating. Of those that died, the operation was usually delayed for several days and the bowel was gangrenous or perforated by this time. In the efferent type there was no relation between mortality and the length of herniated bowel. No patient died with afferent loop herniation causing chronic or recurrent symptoms.

SUMMARY

The salient features of 114 cases have been described because, despite the frequent publication of isolated reports, no review has been made since Himmelmann (1933). The incidence of retro-anastomotic herniation appears to be considerably higher than is generally realized, and although only a few authors have stated the incidence in their cases, the overall figures show an occurrence of 10 in 1168 (0·86 per cent) of afferent loop herniations and 4 in 1340 (0·3 per cent) of efferent type of herniations after partial gastrectomy. Undoubtedly cases have occurred which have not been reported, while many large series of partial gastrectomy operations have not had this complication. Although we have found 158 instances of retro-anastomotic hernia, there were 16 other cases, either mentioned without details or in articles which were not available.

In spite of retro-anastomotic herniation being relatively rare after gastrectomy, nevertheless it should be considered as a possibility in all cases of post-operative small-intestinal obstruction or rupture of the duodenal stump. The afferent loop hernia has been found especially after antecolic partial

gastrectomy, and would appear to add to the mortality of this operation.

The clinical features of the two types have been compared in *Table II*. The course of the condition has sometimes been very rapid. The diagnosis usually has to be made on the clinical features, though rarely help might be obtained from X-rays and pathological investigations. Two features in particular were worthy of stress: firstly the presence of a palpable lump in the left upper abdomen, and secondly the absence of bile in the gastric contents in the afferent loop type.

In carrying out this review we have been surprised by the number of chronic cases, which have accounted for 12·5 per cent of the cases reviewed since Himmelmann (1933), comprising 15 per cent of afferent loop and 11 per cent of the efferent type hernias. The reason for the chronic cases might have been either spontaneous reduction with re-engagement or progressive herniation, while in the afferent loop type there may be an associated kink at the stoma, causing intermittent or incomplete obstruction. Three of the cases of afferent loop hernia had the typical history of post-gastrectomy bile vomiting.

The high mortality from retro-anastomotic hernias could be reduced by prevention, a description of which has not been included in the standard accounts of gastrojejunal anastomosis. Although only a few cases have been reported of herniation through the mesocolon, the suture of the mesocolic edge to the stomach is accepted procedure after retrocolic anastomosis. We would suggest that closure of the hiatus should be performed after retrocolic operations, and that the hiatus should be obliterated as far as possible after antecolic operations.

The high mortality could also be reduced by early operation after the onset of acute symptoms. The chronic or recurrent cases of the efferent type ending in an acute episode also had a high fatality rate, which might be reduced if the possibility were borne in mind during the follow-up of patients with post-gastrectomy syndromes.

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BIBLIOGRAPHY

ARMITAGE, G. (1930), *Brit. J. Surg.*, **18**, 154.
BERKAS, E. M., and FERGUSON, D. J. (1958), *Arch. Surg., Chicago*, **76**, 322.
BORSATO, E. (1949), *Minerva chir.*, **4**, 99.
BORTUZZO, B. (1955), *Friuli med.*, **10**, 51.
BRÉHANT (1947), *Mém. Acad. Chir.*, **73**, 504.
BRYAN, R. C. (1920), *Surg. Gynec. Obstet.*, **30**, 82.
BUCHANAN, E. P. (1932), *Ann. Surg.*, **96**, 359.
BUDDEE (1897), *Dtsch. med. Wschr.*, **23**, 146.
CADENAT (1947), *Mém. Acad. Chir.*, **73**, 525.
CANNON, J. A., and WEEKS, W. H. (1953), *Ann. Surg.*, **138**, 772.
CARICCHIO, C., CABRAL, A. D., GOTO, M., and MAGANO, L. de A. (1952), *Rev. Hosp. Clin., S. Paulo*, **7**, 226.
CASE, T. C. (1958), *Amer. J. Surg.*, **96**, 129.
COLCOCK, B. P. (1954), *Lahey Clin. Bull.*, **8**, 233.
DAVEY, W. W. (1959), *Ann. R. Coll. Surg. Engl.*, **24**, 277.
DENIS, R. (1939), *Lyon chir.*, **36**, 361.
DENOIX, P. (1942), *Pr. méd.*, **50**, 467.
DUBOST, C., and MOREAUX, J. (1956), *Mém. Acad. Chir.*, **82**, 596.
FOWLER, R. S. (1931), *Ann. Surg.*, **94**, 144.
FROMME, A. (1920), *Zbl. Chir.*, **47**, 1505.
HAJDU, N., HARRIS, M. A., and RAMSAY, G. S. (1956), *Brit. J. Radiol.*, **29**, 418.
HAUTEFORT, L. (1931), *Bull. Soc. Chir. Paris*, **23**, 105.
HERBERT, J. J. (1944), *Lyon chir.*, **39**, 83.
HIMMELMANN, W. (1933), *Chirurg.*, **5**, 906.
HOFMANN, A. (1937), *Zbl. Chir.*, **64**, 691.
HOYTE, F. C., JAYNE, W. H. W., and PALLISTER, W. K. (1957), *Lancet*, **1**, 193.
HSÜ, C. H., HU, C. J., and HSÜ, C. M. (1956), *Chin. med. J.*, **74**, 69.
HUBLIN, H. (1951), *Acta chir. scand.*, **101**, 228.
KEENE, R. (1925), *Brit. J. Surg.*, **12**, 791.
KELLING, G. (1900), *Arch. klin. Chir.*, **62**, 1.
KNIGHT, P. R., and BOLT, D. E. (1959), *Brit. J. Surg.*, **46**, 478.
LARSON, E. E. (1930), *Calif. Med.*, **32**, 183.
LECLERC, F. P. (1939), *Lyon chir.*, **36**, 369.
LECLERC, G., and DUBARD (1928), *Ibid.*, **25**, 756.
LEFÈVRE, H. (1947), *Mém. Acad. Chir.*, **73**, 230.
LOCHARD, quoted by ROUX and others (1954).
MCALLISTER, F. F. (1948), *Ann. Surg.*, **128**, 1194.
MASSABUAU, COURTY, BISCAYE, and GUILLAUME (1948), *Pr. méd.*, **56**, 501.
— — — and MARCHAL (1949), *Ibid.*, **57**, 575.
MASSÉ, C. (1955), *Bordeaux chir.*, **2**, 88.
MAYO, C. H., and MAGOUN, J. A. H., jun. (1922), *Arch. Surg., Chicago*, **4**, 324.
MAYO, C. W., STALKER, L. K., and MILLER, J. M. (1941), *Ann. Surg.*, **114**, 875.
MIMPRISS, T. W., and BIRT, St. J. M. C. (1948), *Brit. med. J.*, **2**, 1095.
MOIROUD, M., SALMON, M., and BOUILLON (1938), *Mém. Acad. Chir.*, **64**, 1158.
MONRO, A. K. (1948), *Postgrad. med. J.*, **24**, 177.
MORTON, C. B., ALRICH, E. M., and HILL, L. D. (1955), *Ann. Surg.*, **141**, 759.
OLDHAM, J. B. (1953), *Brit. J. Surg.*, **40**, 625.
OMEZ, Y., CORBEAU, G., and COORNAERT, G. (1950), *Lille chir.*, **5**, 148.
PALUEL (1939), *Lyon chir.*, **36**, 360.
PATRY, R. (1947), *Schweiz. med. Wschr.*, **77**, 571.
PERRY, T., jun. (1954), *Ann. Surg.*, **140**, 119.
PETERSEN, W. (1900), *Arch. klin. Chir.*, **62**, 94.
QUINN, W. F., and GIFFORD, J. H. (1950), *Calif. Med.*, **72**, 18.
REINER, W. C. (1955), *West. J. Surg.*, **63**, 640.
REISCHAUER, quoted by HIMMELMANN (1933).
REYDEN, H. J. van der (1951), *Ned. Tijdschr. Geneesk.*, **95**, 3785.
ROCHET, P. (1939), *Lyon chir.*, **36**, 366.
ROUX, G., NÈGRE, E., and MARCHAL, G. (1954), *J. Chir., Paris*, **70**, 472.
ST. JOHN, F. B., HARVEY, H. D., FERRER, J. M., jun., and SENGSTAKEN, R. W. (1948), *Ann. Surg.*, **128**, 3.
SCHÜRCH, O. (1935), *Dtsch. Z. Chir.*, **244**, 298.
SIMON, M. M. (1956), *Amer. J. Surg.*, **91**, 423.
STAMMERS, F. A. R. (1952), *Brit. J. Surg.*, **40**, 58.
— — (1954), *Ibid.*, **42**, 34.
STANDEVEN, A. (1955), *Ibid.*, **43**, 104.
SUIFFET, W. (1942), *Arch. urug. Med.*, **20**, 371.
SUREN, T. J. J. (1941), *Zbl. Chir.*, **68**, 200.
VERHAEGHE, M. (1949), *Lille chir.*, **4**, 197.
VIANNAY, C. (1938), *Mém. Acad. Chir.*, **64**, 1280.
VULLIET, H. (1918), *Rev. méd. Suisse rom.*, **38**, 672.
WARREN, R. P. (1954), *Ann. Surg.*, **139**, 202.
WEBER, W. (1901), *Beitr. klin. Chir.*, **31**, 240.
WELLS, C. A., and MACPHEE, I. W. (1952), *Lancet*, **2**, 1189.
WEST, J. P. (1953), *Surgery*, **34**, 98.
WOLFSON, A. (1956), *Amer. J. Surg.*, **91**, 298.

CAVERNOUS TRANSFORMATION OF THE PORTAL VEIN

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CAVERNOUS transformation of the portal vein is a not uncommon finding in patients with portal hypertension due to extrahepatic portal obstruction. The condition was first described clearly by Kobrich in 1903, since when many cases have been recorded, the reviews by Klemperer (1928) and Gibson and Richards (1955) being noteworthy.

It is proposed to record briefly the clinical details of 4 further cases before considering the aetiology, pathology, diagnosis, and treatment of the condition.

CASE REPORTS

Case 1.—René T., male, aged 35 years. Recurrent hæmatemeses since July, 1956. Laparotomy in October, 1956, revealed cavernous transformation of the portal vein within the lesser omentum. An attempt to dissect this failed and gastro-enterostomy was performed. Later, the patient was given radiotherapy, presumably in an attempt to shrink the dilated vessels by thrombosis. He continued to bleed intermittently and was referred in December, 1958.

FIG. 205.—*Case* 1. Splenoportogram showing cavernous transformation of portal vein with œsophageal varices. Splenomanometry = 45 cm. water.

FIG. 206.—*Case* 2. Splenoportogram showing similar changes to *Case* 1. Note double splenic vein. Splenomanometry = 32 cm. water.

Apart from his one operation, there was no significant past history. Percutaneous splenomanometry (Dec. 29) showed the portal pressure to be 45 cm. water and splenoportography confirmed that the portal vein ended in a cavernous mass, there being extensive œsophageal varices (*Fig.* 205).

FIRST OPERATION (L. L.) (Jan. 23, 1959).—The upper abdomen was found to be a mass of dense vascular adhesions binding the organs together. Neither splenorenal anastomosis nor splenectomy was possible. The splenic vessels were ligated.

The patient bled again following this operation, the bleeding being controlled by a Leger-Blakemore tube. The latter had to be left in position for periods totalling 150 hr.

SECOND OPERATION (L. L.) (Feb. 5).—Ligation of the œsophageal varices was performed through a left thoracotomy. No bleeding point was seen and an exposure of the fundus of the stomach was known to be impossible. Post-operatively the patient continued to bleed.

He developed an œsophago-pleural fistula and died on Feb. 18.

POST-MORTEM EXAMINATION revealed three features of interest:—

1. The liver appeared normal macroscopically and section confirmed the absence of cirrhosis.

2. The mucosa of the lower œsophagus was ulcerated in several places. The ulcers were sharply defined and section showed granulation tissue at their base, invading the muscular coat. The vessels were dilated and engorged with blood.

3. As found at operation, the liver, spleen, pancreas, stomach, and duodenum were bound together by adhesions to such an extent that their separation was impossible; after fixation, an unsuccessful attempt was made to dissect the mass. Serial sections showed the portal vein to be blocked at its origin by recent thrombus. Around it and continuing into the lesser omentum was a large quantity of dilated thin-walled vessels, few of them having elastic or muscular tissue in their walls. This cavernous tissue was surrounded by inflammatory cells and some fibrosis. Normal nerves were present. The pancreas was normal.

Case 2.—Maria K., female, aged 37 years. This patient was referred from Poland early in 1959. Since 1938 she had suffered from widespread tuberculosis affecting first the sternum, later the lungs, and finally the uterus and adnexæ. In 1954 she was found to be polycythæmic and was treated by venesection and with triethylene melamine.

In 1956 hysterectomy was performed, confirming the tuberculous nature of the salpingitis. Her convalescence was interrupted by the development of a large hæmatoma in the pouch of Douglas, which had to be evacuated on two occasions.

She began to have recurrent hæmatemeses and melæna in 1957, and was found to have an enlarged spleen and œsophageal varices. At the time of her admission her blood-count was 4,820,000 red cells, 13,600 white cells, and 106,000 platelets per c. mm. Splenomanometry (April 14, 1959) 32 cm. water; splenoportography showed

* In receipt of a Ciba Foundation Anglo-French Bursary.

a double splenic vein and the portal vein ending in a cavernous network of vessels (*Fig.* 206).

AT OPERATION (L. L.) (April 23).—The liver appeared normal; biopsy showed minimal fibrosis compatible with early cirrhosis. The lesser omentum was replaced by the cavernous vessels. The splenic vessels were not suitable for splenorenal anastomosis and the enlarged spleen was removed. Section showed it to be fibrotic, there being no evidence of tuberculosis.

Post-operatively she suffered a small pulmonary embolus. The red and white cell counts remained substantially unchanged, but the platelet count rose to 458,000 per c. mm. She has not bled since operation.

Case 3.—Irene T., female, aged 39 years. Following a Cæsarean section in March, 1947, this patient suffered a

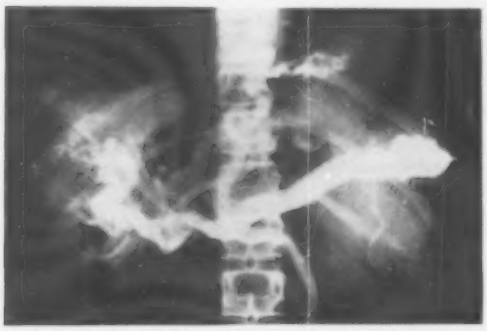

FIG. 207.—*Case* 4. Splenoportogram. Note large splenic vein. Splenomanometry = 25 cm. water.

febrile illness with rigors, and enlarged gall-bladder, but no jaundice. Intravenous cholangiography failed to outline the biliary tract.

Cholecystectomy was performed in September, 1947, the gall-bladder containing calculi. She developed a biliary fistula and the common bile-duct had to be repaired in July, 1948. In January, 1948, she suffered the first of many attacks of hæmatemesis. She was found to have œsophageal varices and an enlarged spleen; splenectomy was performed in December, 1949. After one year bleeding recommenced and has continued at intervals. Since 1956 she has had several attacks of cholangitis.

At the time of her admission to hospital in April, 1959, the liver was found to be slightly enlarged, but there were no other abnormal physical signs. Blood-tests revealed a slight degree of anæmia. The serum-bilirubin was normal, but flocculation tests suggested some impairment of liver function.

AT OPERATION (L. L.) (June 19).—Laparotomy was performed through a low midline incision in order to obtain more information about the state of the portal system. The portal pressure, measured in an ileal vein, was 40 cm. water. Portal venography showed cavernous transformation of the portal vein making its use for anastomosis impossible.

Case 4.—C. M., male, aged 15 years. This child had a suppurative omphalitis in the neonatal period. Since the age of 10 years he had had many hæmatemeses, an œsophageal balloon being used to control them on no less than twenty-six occasions. He was referred in July, 1959, and found to have extensive œsophageal varices with no evidence of cirrhosis. The spleen was enlarged and splenomanometry revealed a pressure of 25 cm. water. Splenoportography (*Fig.* 207) showed cavernous transformation of the portal vein with gross reflux into the left gastric and inferior mesenteric veins.

AT OPERATION (L. L.) (July 17).—The liver was normal. Splenectomy was performed, the splenic vein being 1 cm. in diameter. An anastomosis was fashioned between it and the upper division of a double left renal vein. The child has not bled since the operation.

PATHOLOGY

Remnants of the portal vein can almost always be demonstrated if special stains for elastic tissue are used and serial sections made into the hilum of the liver. The lumen is blocked by organized thrombus in which recanalization frequently occurs. Thrombosis may extend in a retrograde direction to involve tributaries of the portal vein. Mesenteric thrombosis may be a cause of death, as in Klemperer's series, in which 11 of 22 patients were reported as dying from this cause. Dilated veins are found around the portal vein and common bile-duct and in their walls. Some of these have muscle and elastic tissue in their walls and represent accessory portal veins (Sappey, 1883). The walls of others, however, consist only of endothelium and such channels are probably newly formed. Lying in this cavernous tissue are normal nerves and arteries. The hepatic artery itself is often hypertrophied (Ruol, d'Agnolo, and Dal Palu, 1956).

The liver is normal in most cases. Cirrhosis, if present, is seldom severe and may be related to the causative condition (*see* Discussion, *Case* 3).

The spleen is enlarged and shows the changes typical of those present in Banti's syndrome. The splenic vein is occasionally thrombosed and surrounded by cavernous tissue.

Porto-systemic venous communications, including œsophageal varices, are found as in other conditions causing portal hypertension.

AETIOLOGY

The cause of the cavernous changes is debatable. Gibson and Richards (1955) discuss the following three possibilities:—

1. New Formation.—This suggestion was first made by Pick in 1909 and has received some support more recently (Milnes Walker, 1954); however, the pathological findings make it unlikely that this is either an angioma or a hamartoma. The term 'cavernoma' should, therefore, not be used. 'Cavernous transformation' is descriptive, although Hunt (1958) prefers to call the change a "cavernomatous replacement" of the portal vein.

2. Developmental Abnormality.—Congenital stricture of the portal vein is known to occur, and may lead to cavernous transformation at a later state (Auvert, 1958).

An abnormal spread of thrombosis from the ductus venosus and umbilical vein to the portal vein is probably the explanation of the so-called 'congenital cavernoma' (Thompson, 1940). The change is none the less secondary to the portal vein obstruction.

The presence of a formed, albeit thrombosed, portal vein excludes any possibility of an embryological fault in the differentiation of the vitelline plexus.

3. The End-result of Acquired Occlusion.—This is now the generally accepted explanation of cavernous transformation (Auvert, 1953; Ekman, 1957; Gibson and Richards, 1955; Marion, 1953; Parker and Seal, 1955; Milnes Walker, 1959). The

theory is supported by the finding of remnants of the portal vein in the majority of cases in which a special search has been made. Venograms published by Hunt (1958) are suggestive of cavernous transformation developing *pari passu* with extending thrombosis of the portal vein.

The theory explains the wide age incidence of the condition, which may develop at birth, in the neonatal period (*see above*), or at any time thereafter secondary to portal vein thrombosis. Portal vein obstruction is not always associated with cavernous transformation. The reason for this is not known, but may be related to the presence of other portosystemic anastomoses.

The *causes of portal vein thrombosis* are well known and may be classified as follows (Klemperer, 1928):—

a. Compression from Without (e.g., enlarged lymphnodes, carcinoma).

b. Slowing of Portal Circulation.—

i. *General diseases* (e.g., heart failure).

ii. *Local conditions* (e.g., cirrhosis, congenital stricture of the portal vein).

c. Diseases of the Vein Wall including phlebosclerosis, reaction to trauma, pylephlebitis, and spreading thrombosis (neonatal and after splenectomy).

d. Diseases of the Blood (e.g., polycythæmia, leukæmia).

CLINICAL FEATURES

The symptoms and signs are those associated with portal hypertension and hypersplenism. Hæmatemesis and melæna usually cause the patient to seek medical advice. Ascites is rare. Jaundice may be present when there is associated biliary disease or, as in a case reported by Gibson and Richards (1955), when an enlarged anastomotic channel compresses the common bile-duct. Liver function is usually normal. Upper abdominal pain or discomfort may be due either to splenomegaly or to recurrent extension of thrombosis in the portal venous system. The latter may be acute and widespread, causing infarction of the gut.

DIFFERENTIAL DIAGNOSIS

The problem that presents for diagnosis is usually that of hæmatemesis. Clinical examination will reveal an enlarged spleen without the stigmata of cirrhosis. Barium swallow and meal, liver function tests, and a full blood-count are essential, but the most important investigation is splenoportography with splenomanometry (Leger, 1955). This will establish the diagnosis of portal hypertension and, of more importance, will enable the site of the block to be determined. The examination can be extended to include the pyelogram which results from the injection. Splenoportography can be performed as an emergency investigation and should precede barium studies. Having established a diagnosis of portal hypertension, cirrhosis must be excluded. The state of the extrahepatic portal circulation is then assessed from the point of view of a shunt operation.

Extrahepatic obstruction comprises 15–30 per cent of cases in most reported series of portal hypertension (Blakemore, 1951; Hunt, 1958; Linton and Ellis, 1956; Santy and Marion, 1954). In this group the commonest finding is stricture, thrombosis, or cavernous transformation of the portal vein. Pressure on the vein from without by tumour or inflammation is much rarer.

Unfortunately many patients with extrahepatic portal obstruction have already had a splenectomy. The diagnosis of portal hypertension can still usually be made, but details of the state of the portal venous system can only be obtained by operative portal venography.

TREATMENT

The life of these patients is in danger because of hæmorrhage or mesenteric thrombosis. Their liver function is good and for this reason they are much better operative risks than are patients with intrahepatic portal obstruction due to cirrhosis. Many of them are children.

The ideal treatment is to reduce the portal pressure by a venous shunt. In cases of portal thrombosis a portocaval anastomosis may be possible; this is seldom so when cavernous transformation has taken place. Anatomical localization of the block pre-operatively is all-important, for it permits the choice of a left thoraco-abdominal incision, through which all the following operations can be performed:—

1. Splenorenal Anastomosis with preservation of the kidney is obviously the operation of choice. It has a low mortality (0–5 per cent) and the results are good if the splenic vein is of sufficient calibre (8–10 mm. diameter). Unfortunately it may not be possible because of the size of the vein in children or because the vein is atrophic or thrombosed, as after splenectomy. Splenorenal anastomosis is seldom possible under the age of 4 years.

2. Other Venous Anastomoses have been attempted, but the prospect of success is small. The use of a cavernous channel is rarely possible. Some successes have been reported following anastomosis between the mesenteric veins and the vena cava or left renal vein. Venous grafts (Blakemore and Fitzpatrick, 1951) or arterial grafts (Marion, 1958) have been used in association with regional heparinization, but without much success. It may be worth trying one of these lesser shunts in a child while waiting for the splenic vein to increase in size.

3. Splenectomy.—Though removal of the spleen will cure hypersplenism, 80 per cent of patients bleed again afterwards (Whipple, 1951). Splenectomy is usually followed by splenic vein thrombosis which may spread to the mesenteric vessels. Consequently splenorenal anastomosis at a later operation is impossible and the only chance of a cure has been lost.

However, when the splenic vein is itself involved in the cavernous change or is divided into two or three branches, splenectomy may be the only way to relieve the patient of some of his symptoms.

4. Other Treatment.—Emergency ligation of the varices at the lower end of the œsophagus or upper end of the stomach is a useful emergency procedure, as is injection of the varices through an œsophagoscope. If a portosystemic anastomosis is impossible reliance will have to be placed on subtotal gastrectomy (Wangensteen, 1945), œsophagogastrectomy (Phemister and Humphreys, 1947), or gastric transection (Tanner, 1950).

DISCUSSION

The cause of the cavernous transformation is uncertain in *Case* 1. However, this is not surprising since 'silent' pylephlebitis is known to occur following trauma or inflammation in the abdomen. The patient died from hæmorrhage which may have been aggravated by the prolonged use of an œsophageal balloon. Unusually dense adhesions prevented a direct approach to the fundus of the stomach or œsophagogastrectomy. The adhesions may have resulted from the use of radiotherapy after his first operation.

The second patient suffered from abdominal tuberculosis, had a hysterectomy, and was at one time polycythæmic. All these conditions are well known as causes of portal vein thrombosis (Child, 1954).

The third patient almost certainly developed a portal vein thrombosis secondary to inflammation in the lesser omentum. The cavernous changes must have followed or they would have been remarked at her first operation. She presents the typical problem of the post-splenectomy bleeder and, like the second patient, will possibly have to have an œsophagogastrectomy performed later. Her impaired liver function is probably due to recurrent cholangitis.

The child, *Case* 4, is undoubtedly an example of pylephlebitis and thrombosis following neonatal umbilical infection. The condition is probably commoner than is generally recognized (Somers, 1957). Unfortunately the small size of the renal vein in this case may lead to thrombosis of the anastomosis.

SUMMARY

1. The case-histories of 4 patients with cavernous transformation of the portal vein are presented and discussed.

2. It is concluded that this condition results from extrahepatic portal vein obstruction due to a variety of causes.

3. Venous shunt operations, in theory, offer the only prospect of cure, but are often impracticable. In these circumstances, palliation must be attempted by injection of the varices or their removal at open operation.

REFERENCES

AUVERT, J. (1953), *L'Hypertension portale*. Paris: Doin.
— — (1958), *J. Chir., Paris*, **76**, 5, 695.
BLAKEMORE, A. H. (1951), 14. *Congrès de la Société internationale de Chirurgie*. Brussels: Imprimerie Médicale et Scientifique.
— — and FITZPATRICK, H. F. (1951), *Ann. Surg.*, **134**, 420.
CHILD, C. G. (1954), *The Hepatic Circulation and Portal Hypertension*. Philadelphia: Saunders.
EKMAN, C. A. (1957), *Acta chir. scand.*, Suppl. 222.
GIBSON, J., and RICHARDS, R. (1955), *J. Path. Bact.*, **70**, 81.
HUNT, A. H. (1958), *Portal Hypertension*. Edinburgh: Livingstone.
KLEMPERER, P. (1928), *Arch. Path. (Lab. Med.)*, **6**, 353.
KOBRICH (quoted by KLEMPERER) (1903), Inaugural Dissertation, Kiel.
LEGER, L. (1955), *Spleno-portographie*. Paris: Masson.
LINTON, R. R., and ELLIS, D. S. (1956), *J. Amer. med. Ass.*, **160**, 1017.
MARION, P. (1953), *Sem. Hôp. Paris.*, **29**, **2781**.
— — (1958), *J. Chir., Paris*, **76**, 698.
PARKER, R. A., and SEAL, R. M. E. (1955), *J. Path. Bact.*, **70**, 97.
PHEMISTER, D. B., and HUMPHREYS, E. M. (1947), *Ann. Surg.*, **126**, 397.
PICK, L. (1909), *Virchow's Arch.*, **197**, 490.
RUOL, A., D'AGNOLO, B., and DAL PALU, C. (1956), *Acta medica Patavina* (Splene), Suppl. 2.
SANTY, P., and MARION, P. (1954), in *L'Hypertension portale*. Paris: Masson.
SAPPEY, C. (1883), *J. Anat., Paris*, **19**, 517.
SOMERS, K. (1957), *Brit. med. J.*, **2**, 335.
TANNER, N. C. (1950), *Proc. R. Soc. Med.*, **43**, 147.
THOMPSON, W. P. (1940), *Ann. intern. Med.*, **14**, 255.
WALKER, R. M. (1954), *Ann. R. Coll. Surg. Engl.*, **14**, 145.
— — (1959), *The Pathology and Management of Portal Hypertension*. London: Arnold.
WANGENSTEEN, O. (1945), *Canad. med. Ass. J.*, **53**, 309.
WHIPPLE, A. O. (1951), *Ann. Surg.*, **134**, 430.

PHARYNGOGASTRIC ANASTOMOSIS AFTER ŒSOPHAGO-PHARYNGECTOMY FOR CARCINOMA OF THE HYPOPHARYNX AND CERVICAL ŒSOPHAGUS

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THE result of surgical treatment of carcinoma of the hypopharynx and cervical œsophagus is so poor that it should be considered as a form of palliation. Sweet (1954) stated that only in the earliest cases should surgery be considered, and in his series there was not a single 5-year survival. Raven (1952) believed that for some patients surgery may be the only form of treatment, although there may be metastasis in the lymph-nodes. Whether the growth be early or late, the aims of treatment should be a rapid reconstruction of the pharyngogastric continuity. The classical methods advocated and practised by Trotter (1912), Wookey (1940), and Raven (1954) have left much to be desired. Reconstruction of the pharynx with skin tubes is often made difficult by the tendency to form stricture or fistula at the sites of anastomosis. This will require secondary plastic procedures and thus very often it will be months before patients can swallow normally. Occasionally the patient dies of metastasis before the final reconstruction can be completed.

For effective palliation the following conditions must be fulfilled:—

1. It should be a one-stage procedure and deglutition must be effective soon after the operation.

2. The operation should not carry with it a high mortality-rate.

3. The patient must not be worse off than before the operation.

With these aims in view, several methods have been employed to bridge the gap left after excision of the pharynx and upper œsophagus.

1. Use of the Divided Œsophagus.—In 1952 Wooler, by mobilizing the distal end of the divided œsophagus as far as the aortic arch, was successful in anastomosing it to the pharynx. Jack (1955) reported another success in the mobilization and utilization of the lower end of the œsophagus to anastomose it to the divided pharynx. While it is possible to make use of the lower œsophagus, the blood-supply to this segment of the digestive tract must of necessity be precarious as its viability now depends solely on the adequacy and effectiveness of the œsophageal branches of the left gastric artery. As the division of the œsophagus is made farther away from the cardio-œsophageal junction, the danger of avascular necrosis will therefore be greater. Goligher and Robin (1954) questioned the possibility of preserving more than 3–4 in. of the lower œsophagus without running the risk of necrosis.

2. Use of the Jejunum.—The use of the jejunum to replace the œsophagus has been well tried and is a very useful means of restoring œsophago-gastric continuity. The success of its use by Yudin (1944) for œsophageal stricture has led many to use the jejunum. While it is a very suitable procedure for replacing the œsophagus up to the level of the lower part of the neck, stretching it to the pharynx has a much greater chance of inducing necrosis. Allison (1959) nevertheless succeeded in fashioning a Roux loop and anastomosing one end to the base of the tongue and oropharynx. The lower end was inserted into the stomach. There is, however, a great deal of difficulty connected with its use. The variation frequently found in the blood-supply of the jejunum sometimes precludes its use. Often the vascular arcades may be far from the mesenteric edge of the intestine, and when this is the case the length of the jejunum that can be mobilized will be limited.

3. Use of the Colon.—For lesions of the œsophagus the colon has been extensively used either to replace or by-pass it. Kelling (quoted by Yudin, 1944) made use of the transverse colon to replace the œsophagus isoperistaltically, while Vulliet (also quoted by Yudin, 1944) did it in a reverse direction. Sherman, Mahoney, Dale, and Stabins (1955) utilized the right side of the terminal ilium for œsophagoplasty. Since then it has been popular and extensively used in the United States (Nadal and Gustavson, 1957; Battersby and Moore, 1959). Goligher and Robin (1954) reported a case in which they used the left side of the colon and through a subcutaneous tunnel anastomosed it antiperistaltically to the stomach and the pharynx. There is very good blood-supply via the left branch of the middle colic artery to this segment of the colon for its viability. However, any antiperistaltic anastomosis tends to drive the contents of the stomach or jejunum towards the pharynx. This causes a great deal of discomfort and annoyance to the patient.

4. Use of the Stomach.—Kirschner (quoted by Yudin, 1944), through a subcutaneous tunnel, anastomosed the fundus of the whole stomach to the cervical œsophagus. He then dealt with the divided cardiac end of the œsophagus by anastomosing to the jejunum. This procedure is hazardous because of the extra anastomosis of the lower end of the œsophagus to the jejunum. Meyers (1912) advocated that a Jianu tube be constructed and then anastomosed to the exteriorized cervical œsophagus. Heimlich and Winfield (1955) extended this procedure by performing a reversed gastric tube along the greater curvature from just proximal to the pylorus almost to the fundus of the stomach. After removing the spleen close to the hilum, they mobilized the tail of the pancreas as far as the aorta and then anastomosed the pyloric end to either the pharynx or cervical œsophagus. Heimlich and Gavriliu, at a meeting in Mexico (1957), reported a total of 61 cases operated thus, and, of these, 5 developed fistula at the œsophagogastric suture line. Two of the 61 subsequently developed stricture at the site of anastomosis. Two of our cases operated in this manner developed leakage, but both healed spontaneously. Although Goligher and Robin (1954) doubted the feasibility of suturing the fundus of the stomach to the pharynx, we have successfully operated on 3 consecutive cases without any death.

TECHNIQUE OF PHARYNGOGASTRIC ANASTOMOSIS

Anæsthesia.—All 3 cases were operated upon under anæsthesia of pentothal, nitrous oxide, and oxygen, with curare as a relaxant.

Position of Patient.—The position of the patient was supine throughout the procedure, with the right arm in abduction.

Operation.—The skin of the neck, chest, and abdomen were prepared and draped. Exploration of the neck was done first. A U-shaped incision was made with one limb extending from the right mastoid process and coursing downwards across the middle of the anterior border of the right sternomastoid muscle to reach about 1 in. above the suprasternal notch. The other limb of the U was made from the left mastoid process to the level corresponding to the lower limit of the right limb. The lower ends were then connected by a transverse incision.

The platysma was reflected with the skin and this was carried as high up as the mandible. Later it was found that a long collar incision would give as good an exposure as a U-shaped one.

After dividing the investing fascia along the anterior border of the left sternomastoid muscle, the left carotid sheath was exposed. This allowed examination for any metastasis in the lymph-nodes. Similarly the right carotid sheath was exposed and palpation for metastasis was carried out. On dividing the left omohyoid as low down as possible, the thyroid vessels were divided between ligatures. The growth in the œsophagus or pharynx was next palpated for extension on the left side. By a similar manœuvre the right side was also examined. By blunt dissection the trachea and œsophagus could be lifted from the prevertebral fascia. If there was no infiltration into the prevertebral fascia, block dissection was proceeded with by removal of the sternomastoid and internal jugular vein on the side of the growth while preserving the internal jugular vein and the sternomastoid of the opposite side. If necessary the opposite sterno-mastoid could be divided completely and then re-sutured at the end of the surgical extirpation. The sternohyoid and sternothyroid were next divided as low as possible. The isthmus of the thyroid gland was then divided between clamps, ligatured, and the whole gland dissected free from the trachea and larynx. Extension of the growth into the thyroid sometimes

occurs, as in *Case* 1, and in which case removal of the involved lobe of the thyroid would have to be carried out. It is desirable to preserve one lobe of the thyroid with the parathyroids of that side, but one should not compromise their preservation at the expense of adequate surgery.

Division of the trachea was made at the level of the third or fourth tracheal ring, depending on the level of the growth. After dividing the anterior wall of the trachea the endotracheal tube was removed. A short-cuffed tube, previously sterilized, was inserted

FIG. 208.—*Case* 1. Showing patient at the time of admission.

into the opening created by the division of the anterior tracheal wall. The posterior wall of the trachea was then dissected free from the œsophagus. A circular piece of skin about 1 in. in diameter was excised in the suprasternal fossa. The posterior wall of the trachea was divided. The cuffed tracheal tube was removed and the trachea brought out through the circular incision in the suprasternal fossa. After suturing the divided trachea to the circular skin edge, the cuffed tube was re-inserted and the anæsthesia continued. This could be done very quickly without disturbing the patient.

If the growth was high the hyoid bone was removed with it, but if it were low the division was made through the thyrohyoid membrane after dividing the infrahyoid muscles. The posterior pharyngeal wall was removed at least 3 cm. from the gross margin of the lesion. The pharynx, larynx, and the mass of dissected tissue was left attached only to the œsophagus. This mass of tissue containing the tumour was enclosed in a sterile glove and securely tied. The œsophagus was next dissected as low down as possible into the superior mediastinum. The skin-flap, which had been protected by a moist pack all along, was further protected with new towels and the operation on the abdomen was now carried out.

Mobilization of the Stomach.—The abdomen was entered through a long supra-umbilical midline incision. The stomach was first mobilized along the greater curvature by dividing the gastrocolic omentum right to the origin of the right gastro-epiploic artery. Great care must be taken not to damage the arterial arcade along the greater curvature. The vasa brevia were individually ligatured and divided. The lesser curvature was mobilized by cutting the left gastric vessels separately between ligatures. The lesser omentum was then just cut through with a pair of scissors. The lower end of the œsophagus was freed

FIG. 209.—*Case* 1. Radiograph after instillation of lipiodol.

FIG. 210.—*Case* 1. The same patient eating solid food two weeks after the operation.

by dividing the vagi, and by finger dissection this procedure could be extended into the thorax. The second part of the duodenum with the head of the pancreas was mobilized as far as the inferior vena cava. The abdomen was then closed.

Dissection of the Thoracic Œsophagus.—A right anterior thoracotomy was made through the sixth

intercostal space. The vena azygos was divided between ligatures. The œsophagus was then dissected free from the lower end through to the root of the neck. If the dissection of the œsophagus in the neck had been adequate this part of the operation could be easily carried out. The stomach was delivered into the thorax by gentle traction on the œsophagus.

FIG. 211.—*Case* 1. Post-operative barium examination showing the stomach extending to the pharynx. The gastric rugæ were straightened out.

The cardio-œsophageal junction was severed and the cardia closed. With stout linen thread the fundus of the stomach was anchored to the divided end of the œsophagus. Traction on the cervical œsophagus would now bring the stomach into the neck. After fixing the stomach in the chest so that there would be no rotation, the chest was closed with underwater seal.

Anastomosis in the Neck.—It was found that the stomach would stretch as high as the chin. The mass of tissue with the larynx, pharynx, and the whole of the œsophagus was now removed after cutting through the linen thread. The anastomosis between the stomach and pharynx was made with an outer sero-musculo-muscular layer and an inner through-and-through layer of interrupted silk. The neck was closed with two drains in the supraclavicular fossæ.

Blood was given throughout the operation. There was a drop of blood-pressure of 20–40 mm. Hg in each case during the 'pull through' of the stomach into the neck. However, this quickly recovered at the time of anastomosis.

CASE REPORTS

Case 1.—Kowloon Hospital No. 6595/58. Mrs. T. B., aged 68 years, was admitted on Aug. 28, 1958, with a history of progressive dysphagia for nine months. She could feel the obstruction in the neck and three months after the onset she could swallow only fluids.

ON EXAMINATION.—She was found to be very frail (*Fig.* 208) and her body-weight was only 69 lb. She had œdema of both ankles. Her respiratory and cardio-vascular systems revealed no abnormality except for thickened vessels. Her blood-pressure was 100/60 mm. Hg. Except for an enlarged firm lymph-node in the left side of the neck, there was no evidence of distant metastasis.

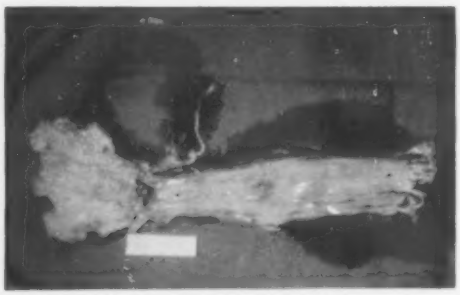

FIG. 212.—*Case* 1. Showing the operation specimen.

Laboratory Findings.—Hæmoglobin 51 per cent; Albumin 3·9 mg. per cent; Globulin 2·5 mg. per cent.

Barium Swallow.—Barium swallow was unsuccessful as she could not swallow the medium. It was repeated with a small amount of lipiodol which could just outline the irregularity in the post-cricoid region. A bulging soft-tissue shadow could be seen just below this irregularity (*Fig.* 209).

Œsophagoscopy.—An ulcerative growth could be seen in the post-cricoid region extending into the laryngo-pharynx. Biopsy revealed a poorly differentiated squamous-celled carcinoma.

As she was swallowing liquid with difficulty and was anæmic, she was given a pint of blood. After a further few days of intravenous therapy, she was strong enough to swallow liquids. Milk reinforced with digested protein was given to her liberally. She was encouraged to get out of bed, and at the end of two weeks her condition had improved considerably. Her œdema had by this time disappeared. Her hæmoglobulin was 71 per cent, albumin 4·1 mg. per cent, and globulin 2·2 mg. per cent.

Since her general condition was judged to be at its best, she was operated upon on Sept. 11.

OPERATIVE FINDINGS.—The growth was found to be from the post-cricoid region and to have infiltrated both lobes of the thyroid gland. However, it was still free from the prevertebral fascia. It was therefore decided to proceed with the resection as planned. The pharynx, larynx, œsophagus, and thyroid were removed and, in addition, a block dissection of the neck on the left side was carried out. The abdominal part of the operation was done without any difficulty. Thoracotomy was done and the stomach delivered into the chest. As the stomach was being drawn into the neck, the blood-pressure dropped to 90 mm. Hg, but it quickly rose to its previous level after all manipulation was stopped for a few minutes. The anastomosis was completed without any difficulty and there was no tension at the suture line.

POST-OPERATIVE COURSE.—At the end of the operation her blood-pressure was 110/80 mm. Hg. She returned to the ward in good condition. She received a total of 3 pints of whole blood; 2 pints were given during the operation and another 1 pint was given post-operatively. On the third post-operative day she developed tetany. This was controlled by intravenous calcium gluconate. There were three subsequent attacks during the next two weeks, and at each attack calcium gluconate was given. Oral calcium with vitamin A was given simultaneously. During the

attacks of tetany the calcium estimation varied from 5·6 mg. per cent to 7·6 mg. per cent.

Oral feeding was started seventy-two hours following the operation and she could swallow fluid without difficulty. By the end of the first post-operative week she was able to eat solid food (*Fig.* 210). Regurgitation of gastric juice took place whenever she was in a state of agitation, but this gradually disappeared after a few weeks.

FIG. 213.—*Case* 2. Showing the patient at the time of admission.

From the third post-operative day there was a great tendency to encrustation at the tracheotomy opening. On removing the crust it was found that the tracheal opening had undergone necrosis. This subsequently healed with stricture formation and a refashioning of the opening was necessary.

There was accumulation of serum in the neck after the drains had been removed. This stopped spontaneously when the accumulated serum had been aspirated.

Post-operative Barium Swallow (*Fig.* 211).—The stomach was in the form of a tube and the anastomosis to the pharynx could be made out at the level of the hyoid bone.

PATHOLOGICAL SPECIMEN (*Fig.* 212).—Consisted of 24 cm. of the œsophagus with the larynx and about 2·5 cm. of trachea. At the level of the larynx in the upper part of the œsophagus there was a neoplastic growth measuring 70 mm., extending through the whole œsophageal circumference. This growth was verrucous and ulcerated. Histology of the growth showed poorly differentiated squamous-celled carcinoma.

She was discharged from hospital on Dec. 17. Her body-weight was now 78 lb. and she could eat any food given to her.

FOLLOW-UP.—She had been well since her discharge from hospital until May, 1959, when she started to have repeated vomiting. Examination revealed that she was extremely emaciated. There was ascites and hard nodules could be felt in the liver. She died shortly after admission to another hospital. No post-mortem examination was carried out.

Case 2.—Kowloon Hospital No. 7814/58. Mr. F. C., aged 39 years, a mortuary attendant, was admitted on Oct. 8, 1958, because of progressive dysphagia for three months. One year prior to admission he experienced some pain and discomfort in the throat. This was followed by difficulty in swallowing solids and he was living on semi-solid food. By the time he sought medical aid, he could take only liquid slowly and with difficulty. He had lost 10 lb. in weight within this period.

ON EXAMINATION.—He was a well-developed man who showed signs of recent loss of weight (*Fig.* 213). However, he appeared fit. A mass was palpable behind the thyroid and cricoid cartilages. It was freely movable from side to side and was hard in consistency. There were no palpable lymph-nodes. No other abnormality was detected on further physical examination.

Laboratory Findings.—Hæmoglobin 82 per cent; Hæmatocrit 37 per cent; Albumin 4·1 mg. per cent; Globulin 2·0 mg. per cent.

Barium Swallow.—This showed a hold-up in the region of the pyriform fossa (*Fig.* 214).

FIG. 214.—*Case* 2. Barium examination showing the retention of barium at the pyriform fossa.

FIG. 215.—*Case* 2. Post-operative swallow showing the pharyngogastric anastomosis.

Œsophagoscopy.—There was an ulcerative growth extending from the left pyriform fossa into the post-cricoid region. Biopsy revealed squamous-celled carcinoma.

OPERATIVE FINDINGS.—A long collar incision was made in this case. It extended from the lateral border of one trapezius to the other. The growth was found to be freely

FIG. 216.—*Case* 2. Operation specimen. The whole œsophagus with the growth extending into the left pyriform fossa.

movable. There were shotty lymph-nodes extending all the way down to the superior mediastinum. The pharynx, larynx, and œsophagus were removed together with a block dissection on the left side of the neck. The patient stood the operation very well and the lowest systolic blood-pressure recording during the 'pull through' of the stomach into the neck was 100 mm. Hg.

POST-OPERATIVE COURSE.—His post-operative course was smooth throughout. Two pints of whole blood were given to him and for the next three days he was put on intravenous fluids. The suture line between the pharynx and stomach was examined on the third post-operative day and was found to be healing well. He was given oral fluids after the examination. For the next two days he had regurgitation of gastric contents, but this disappeared after he was reassured that it would not recur. By the end of the week he was eating normal diet. On the eighth day after operation he was walking about the ward.

FIG. 218.—*Case* 3. The patient at the time of admission.

Post-operative Barium Swallow (*Fig*. 215).—The barium passed through the stomach easily. The site of the anastomosis was at the level of the hyoid bone.

He was discharged on the eighteenth post-operative day and appeared well (*Fig*. 217). However, on Dec. 30 he was admitted again because of breakdown of wound in the neck. Examination revealed induration extending down into the root of the neck. Within seven days he had become emaciated and died suddenly on Jan. 7, 1959. Post-mortem examination was refused.

FIG. 217.—*Case* 2. The patient 18 days after the operation.

THE OPERATION SPECIMEN.—The specimen was the œsophagus together with the whole larynx. The œsophagus measured 19 cm. in length. Cross-section of the lower end of the amputation showed nothing remarkable, but the posterior and lateral pharyngeal wall showed an

FIG. 219.—*Case* 3. Pre-operative barium examination. Note the filling defects at the level of the 6th and 7th cervical vertebræ.

ulcerating fungating growth which measured roughly 4 cm. in diameter on the left and 2 cm. in diameter on the right. The laryngeal mucosa was œdematous (*Fig*. 216).

Microscopically, the lining epithelium had partly disappeared and was replaced by sheets of anaplastic epithelial cells. Among the epithelial cells there was normal glandular tissue. In one of the lymph-nodes examined the normal architecture was distorted. There were no lymph-follicles or germinal centres found.

Case 3.—Kowloon Hospital No. 1288/59. Mr. L. F., aged 39 years, a scavenger coolie, was admitted on Jan. 4, 1959, with the complaint of pain and discomfort in the neck for nine months. He could at first take solid food, but gradually he could take only liquid food. He lost some weight, but the amount was unknown to him.

FIG. 220.—*Case* 3. Showing the patient taking normal diet 10 days after the operation.

FIG. 222.—*Case* 3. Showing the operation specimen. Note the extent of the growth.

ON EXAMINATION.—A well-developed and well-nourished man who appeared rather lethargic and was slow mentally (*Fig.* 218). He could swallow water with difficulty. A bulge was palpable just behind the cricoid cartilage. No lymph-nodes were palpable in the neck. No other abnormality was detected on careful physical examination. His blood-pressure was 120/80 mm. Hg. His body-weight was 98 lb.

Laboratory Findings.—Hæmoglobin 95 per cent; Albumin 4·6 mg. per cent; Globulin 2·4 mg. per cent.

Barium Examination.—There was a hold-up of the barium and a filling defect could be made out from the lower border of the fifth cervical vertebra to the upper border of the seventh cervical vertebra. The trachea was slightly displaced forward by growth at this level. (*Fig.* 219.)

Œsophagoscopy.—An ulcerative growth was found in the post-cricoid region extending into the hypopharynx. Biopsy showed that it was an epidermoid carcinoma.

AT OPERATION.—The growth was found to have extended beyond the pharyngo-œsophageal wall and to have invaded the right lobe of the thyroid gland. Posteriorly the prevertebral fascia was free from infiltration. Lymph-nodes were palpable along the right carotid sheath down to and behind the right sternoclavicular joint. Radical excision of the pharynx, larynx, and whole œsophagus, with block dissection of the neck on the right side and right lobe of the thyroid, was carried out. When the stomach was withdrawn into the neck the blood-pressure dropped to 70 mm. Hg and then became imperceptible. However, anastomosis was completed and at the time of closure the blood-pressure had risen to 90/50 mm. Hg. Two pints of whole blood were given at the time of operation and a further 2 pints were given during the next 12 hr.

FIG. 221.—*Case* 3. Post-operative barium examination.

FIG. 223.—*Case* 3. The patient at the completion of course of post-operative irradiation.

POST-OPERATIVE COURSE.—He recovered from the shock quickly. Feeding was started after 72 hours. From then on his convalescence was uneventful. Apart from minor infection of the wound in the neck, he was quite comfortable and was able to take normal diet 10 days after the operation. (*Fig.* 220.)

A course of nitrogen mustard was given, starting from the second post-operative day. He took it very well and had no reaction.

Three weeks after the operation he weighed only 88 lb. and his ability to gain weight was slow. A course of irradiation was given after the operation, as it was felt that the growth had been extensive and distant metastasis had probably taken place.

Post-operative Barium Examination.—The barium passed through the stomach without difficulty. The site of anastomosis was at the level of the hyoid bone (*Fig.* 221).

FOLLOW-UP.—He went through the course of irradiation with minimal amount of discomfort and was discharged at the end of his treatment (*Fig.* 223). At this time he weighed only 95 lb. When last seen in July, 1959, he weighed 108 lb. and appeared fit with no sign of recurrence or metastasis.

OPERATED SPECIMEN.—Consisted of 14 cm. of the œsophagus together with the larynx and upper part of the trachea. Involving the upper portion of the œsophagus and lower portion of the larynx, but not the margins of the vocal cord, was a diffuse area of erosion 6 cm. long, extending over the entire circumference of the viscus. Underlying this there were diffuse thickening and infiltration by whitish tissue. There was no gross extension into the trachea. The right lobe of the thyroid was attached to the specimen, as were fragments of skeletal muscle and adipose tissue. (*Fig.* 222.)

Histologically it was composed of very cellular tissue covered by necrotic material. Sheets of epithelial cells infiltrated the underlying tissue. There were numerous epithelial pearls.

DISCUSSION

The problems of carcinoma of the cervical œsophagus and the hypopharynx are difficult to solve and the prognosis is even worse. The 5-year survival rate is extremely poor. As the life span of these unfortunate patients is short, any treatment should not be too prolonged and attempts must be made to enable them to lead a life as comfortable as possible.

Hitherto many methods have been advocated and used, but there is not a single procedure that is not fraught with danger and complications. Although Goligher and Robin (1954) doubted the possibility of using the fundus of the stomach for anastomosis to the pharynx, it is a feasible method.

Advantages of using the Stomach.—1. The blood-supply to the stomach is the best of all the portions of the gastro-intestinal tract that have been used to replace the œsophagus. After mobilization, it still has two large arterial arcades, with the main feeding vessels as the right gastric and the right gastro-epiploic arteries. If the main arteries are not damaged, any part of the stomach will be richly supplied with blood.

2. The stomach is very elastic. It is amazing to what length it will stretch. Because of the elasticity of the stomach, Tanner (1954) advocated that one-third to one-half of the width of the divided stomach be used for anastomosis in performing a Polya gastrectomy.

3. Healing power of the stomach is very good. It healed well in each of the 3 cases. There was no anxiety as far as leakage was concerned. The suture line could be examined by depressing the tongue.

Distance that Stomach has to traverse to reach the Neck.—The shortest distance for the stomach to reach the neck is through the thorax. For the stomach which is an organ attached to the posterior abdominal wall to pass through a subcutaneous tunnel it has to traverse an extra distance of at least 1 in. The length of the stomach could be fully utilized by mobilizing the duodenum with the head of the pancreas as far as the inferior vena cava. This gives the stomach extra length, as the duodenum would be lying on the œsophageal hiatus when the stomach is drawn upwards.

Severity of the Operation.—That the operation is severe is beyond doubt. It involves extensive dissection in the neck, with opening and closure of both the abdominal and thoracic cavities. However, all 3 patients, one of whom (*Case* 1) was a frail emaciated old woman, stood the various stages of the operation very well, except at the time when the stomach was being drawn through the thorax into the neck. *Case* 3 went into severe shock mainly because the mobilization of the œsophagus in the neck was inadequately done. The dissection in the thorax with opening and closure took slightly more than half an hour. If two teams were to work simultaneously the operating time could be cut down by at least half. As it was, each case took from four to four and a half hours. Only one team was working at a time.

SUMMARY

The surgical treatment of carcinoma of the cervical œsophagus and hypopharynx is far from being satisfactory. Multiple-staged operations should, whenever possible, be avoided. Deglutition should be restored as soon as possible. Three cases in which the fundus of the stomach was anastomosed to the pharynx are reported. The operation is described in detail. Although the operation is very severe, all 3 cases did well. The post-operative course was smooth and healing was very good.

We wish to thank the Hon. Director of Medical and Health Services for permission to publish these cases and for the use of the records. To Professor P. C. Hou, of Hong Kong University, who examined the pathological specimens, we owe much for his kind advice and help.

REFERENCES

ALLISON, R. (1959), *Proc. R. Soc. Med.*, **52**, 176.
BATTERSBY, J. S., and MOORE, T. C. (1959), *Surg. Gynec. Obstet.*, **109**, 207.
GOLIGHER, J. C., and ROBIN, I. G. (1954), *Brit. J. Surg.*, **42**, 283.
HEIMLICH, H. J., and GAVRILIA (1959). Personal communication.
— — and WINFIELD, J. M. (1955), *Surgery*, **37**, 549.
JACK, G. D. (1955), *Brit. J. Surg.*, **42**, 530.
MEYERS, W. (1912), *J. Amer. med. Ass.*, **62**, 100.
NADAL, J. W., and GUSTAVSON, R. G. (1957), *Arch. Surg.*, **74**, 442.
RAVEN, R. W. (1952), *Brit. med. J.*, **1**, 951.
— — (1954), *Brit. J. Surg.*, **42**, 113.
SHERMAN, C. D., jun., MAHONEY, E. B., DALE, W. A., and STABINS, S. J. (1955), *Cancer*, **8**, 1198.
SWEET, R. H. (1954), *J. Amer. med. Ass.*, **155**, 422.
TANNER, N. C. (1954), *Postgrad. med. J.*, **30**, 457.
TROTTER, W. (1913), *Lancet*, **1**, 1075.
WOOKEY, H. (1940), *Brit. J. Surg.*, **27**, 696.
WOOLER, G. (1952), *Proc. R. Soc. Med.*, **45**, 264.
YUDIN, S. (1944), *Surg. Gynec. Obstet.*, **78**, 561.

LIME-WATER BILE

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THE term 'lime-water bile' was first used by R. Volkmann in 1908 to describe a thick opaque type of bile. In 1911 Churchman found a putty-like material in the gall-bladder in a patient who had suffered from acute cholecystitis and in whom the cystic duct was obstructed. J. Volkmann in 1926 reported 2 cases in which the gall-bladder contained a white opaque fluid, and he coined the term 'milk of calcium bile'. Knutson (1933) replaced this with the term 'limy bile', as the amorphous material in the gall-bladder was not always a milky fluid but often a semi-solid paste and sometimes a solid concretion. This last term is the one most commonly used in American literature.

The condition lime-water bile is due to the formation of a suspension of crystals of calcium salts in the gall-bladder. These salts render the contained bile opaque to X rays and thus give the characteristic feature of the condition, the delineation of the affected part of the biliary tract on plain films. A search through the literature suggests that the condition is a rare one, as we were able to trace only 58 recorded cases; but it is possible that many others were diagnosed as 'calcification of the gall-bladder' or were missed because preliminary films had not been taken before cholecystography.

In all but 3 of the previously reported cases the radio-opaque bile was confined to the gall-bladder. In these 3 cases (Cohen, 1 case, 1955; Simmonds, 2 cases, 1957) the common bile-duct was visualized as well as the gall-bladder.

We have recently encountered 3 more cases of the latter type, and their study forms the subject of this paper.

CASE REPORTS

Case 1.—Mrs. R., aged 43 years, was admitted to hospital after a week's attack of severe epigastric pain. Four days before admission she became jaundiced, with pale stools and dark urine. Plain X-ray films which were taken of the abdomen (*Fig.* 224) showed fine amorphous calcified material in the region of the fundus of the gall-bladder and further deposits of similar material outlining the common bile-duct. A densely calcified stone, 0·75 cm. in diameter, was present at the lower end of the duct. The gall-bladder failed to visualize after the ingestion of an oral dose of contrast medium.

Biochemical investigations were carried out four days before operation, with the following findings: Serum-bilirubin 2·4 mg./100 ml.; Alkaline phosphatase 24 units; Zinc turbidity 3 units; Thymol turbidity 1 unit; Plasma albumin 3·3 g./100 ml.; Plasma globulin 3·1 g./100 ml.

These findings are in keeping with the clinical picture of obstructive jaundice.

At Operation (Nov. 18, 1958).—Mr. D. McIntosh found that the gall-bladder and common bile-duct contained a yellow pultaceous material. The common duct was obstructed by two small gall-stones. These were removed, the duct irrigated, and its patency confirmed by passing bougies. The gall-bladder was removed and the common duct closed around a T-tube. The patient made an uneventful recovery. The T-tube draining the common bile-duct was removed on the eleventh post-operative day, and the patient has remained well. Plain X-ray films taken two weeks after operation showed no remaining radio-opaque material in the abdomen.

The Specimen.—The gall-bladder was 8 cm. in length. The proximal part was very narrow and measured only 1 cm. in diameter. The fundus, however, was more normal in appearance. The mucous membrane was intact and its appearance suggested a slight cholesterolosis. The wall was thickened owing to fibrosis, inflammatory reaction, and muscular hypertrophy (Dr. J. D. McGregor pathologist).

Fig. 224.—*Case* 1. Prone film demonstrating limy bile present in the fundus of the gall-bladder and also outlining the lower end of the common duct. Note the stone formation present in the lower end of the duct (arrowed).

Case 2.—Miss F., aged 36 years, began to complain of aching pain in the right hypochondrium five weeks before admission. This pain lasted for two weeks and was accompanied by frequent attacks of vomiting. Three days before admission she had a recurrence of the same type of pain together with further vomiting. For three weeks before admission she had noticed that the stools were pale and the urine dark in colour, but she had not noticed any change in the colour of her skin. There were no other relevant symptoms. Some ten years previously she had been treated for pulmonary tuberculosis, but there had been no recent activity.

Plain X-ray films of the abdomen (Nov. 28, 1958) (*Fig.* 225) revealed a calcified opacity in the right hypochondrium which had the appearance of a stone in the lower end of the common bile-duct. Proximal to this stone the common bile-duct, which was grossly dilated, was outlined by a radio-opaque material having the appearance of lime-water bile. In the fundus of the gall-bladder there was a quantity of biliary sand which in the erect position formed a horizontal level.

On Admission (Dec. 14).—There was an icteric tinge in the conjunctivæ. Slight tenderness was elicited over the gall-bladder. A second series of plain X-ray films was taken Dec. 14 (*Fig.* 226) and this showed appearances similar to those previously described with the addition that reflux of the lime-water bile had occurred along the pancreatic duct.

AT OPERATION (Dec. 15).—The gall-bladder and common bile-duct were found to be filled with a pale yellow material and the common duct was markedly distended. The pancreas was tense and œdematous. The common bile-duct was opened and the contained semi-solid material

FIG. 225.—*Case* 2. Demonstrating a grossly dilated common duct filled with bile, with stone formation present at the lower end and a further quantity of limy bile present in the fundus of the gall-bladder.

was removed by irrigation. The obstructing stone was impacted at the ampulla of Vater and had to be removed by the transduodenal route. The common bile-duct was closed around a T-tube. In view of the state of the pancreas, it was decided not to remove the gall-bladder, but simply to evacuate its contents and drain it.

A post-operative cholangiogram on Jan. 3, 1959 showed a free flow of the contrast medium from the common duct into the duodenum. A small diverticulum was noted at the lower end of the common bile-duct, presumably at the site of lodgement of the duct stone; the size of the common bile-duct had shrunk considerably.

The drain in the gall-bladder was removed on Dec. 24, 1958, and on Jan. 7, 1959 the T-tube was removed from the common bile-duct. The patient did well at first. Subsequently she developed a respiratory illness which was found to be a reactivation of her previous tuberculous focus in the left lung. This responded very well to appropriate chemotherapy, and she returned home on May 30. A small fistula had persisted in the abdominal wall leading to the gall-bladder, and continued discharging a trace of bile and pus from time to time. When last seen on June 25 the opening was very small and tending to dry up for days at a time.

BIOCHEMICAL INVESTIGATIONS were carried out, and the following results obtained:—

Blood Chemistry.—

1. On the 7th post-operative day, serum-calcium was 10·1 mg. per cent.

2. On the 24th post-operative day the findings were: Serum-calcium 11·0 mg./100 ml.; Serum-bilirubin 0·6 mg./100 ml.; Alkaline phosphatase 21 units; Zinc turbidity 1 unit; Thymol turbidity 1 unit; Total cholesterol 245 mg./100 ml.; Free cholesterol 55 mg./100 ml.; Serum inorganic phosphorus 4·9 mg./100 ml.

Urine Chemistry.—On low-calcium diet (130–150 mg./day): Twenty-four-hour calcium excretion 82 mg.; Twenty-four-hour creatinine excretion 660 mg.; Calcium/creatinine ratio 0·12.

These findings show that the calcium metabolism is within normal limits.

Case 3.—Mrs. S., aged 57 years, had complained of flatulent dyspepsia for a year. Three weeks before admission she had a single attack of biliary colic lasting

FIG. 226.—*Case* 2. Demonstrating reflux of limy bile along the pancreatic duct (arrowed).

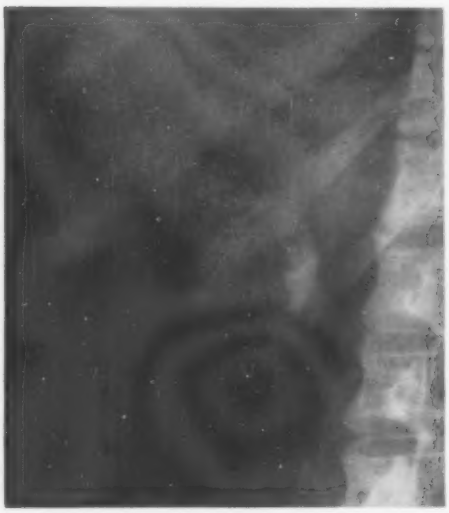

FIG. 227.—*Case* 3. Demonstrating the lower end of the common duct and numerous small radio-opaque stones present in the fundus of the gall-bladder and in the common duct.

half an hour. One week later she developed jaundice with pale stools and dark urine. This persisted until the time of admission. Plain X-ray films of the abdomen (*Fig.* 227) showed numerous opaque and mixed calculi within the gall-bladder and cystic and common bile-ducts. There was also a quantity of opaque material in the ducts and gall-bladder. Following the injection of biligrafin none of the contrast medium appeared in either gall-bladder or ducts up to two hours.

AT OPERATION (June 12).—Mr. J. R. Cameron found that the gall-bladder was thickened and contained a number of stones. The common bile-duct, which was also thickened, was opened and found to contain a quantity of semi-solid material together with one stone. These were removed and the duct washed out. Operative cholangiography showed no residual stones and a bougie passed easily into the duodenum, confirming the patency of the common duct. The gall-bladder was removed and the common duct closed around a T-tube.

The gall-bladder was 8·5 cm. in length. It contained thick, dark-green bile in which were several small, hard pigment stones. The mucosa was granular. The wall showed no evidence of fibrosis, but was œdematous and infiltrated with polymorphonuclear leucocytes.

The patient made a satisfactory recovery from the operation and the jaundice had disappeared by the seventeenth day after operation.

BIOCHEMICAL INVESTIGATIONS gave the following results:—

Nine days before operation: Serum-bilirubin 4·6 mg./100 ml.; Alkaline phosphatase 25 units; Zinc turbidity 4 units; Thymol turbidity 1 unit; Prothrombin activity 40 per cent; Plasma albumin 4·5 g./100 ml.; Plasma globulin 2·7 g./100 ml.

Five days after operation: Serum-bilirubin 3·7 mg./100 ml.; Alkaline phosphatase 20 units; Zinc turbidity 4 units; Thymol turbidity 2 units.

Eighteen days after operation: Serum-bilirubin 1·0 mg./100 ml.; Alkaline phosphatase 20 units; Zinc turbidity 3 units; Thymol turbidity 3 units; Plasma albumin 4·6 g./100 ml.; Plasma globulin 1·6 g./100 ml.

These figures are in keeping with the clinical picture of obstructive jaundice relieved by operation, the levels having not quite returned to normal at the time of the last investigation.

DISCUSSION

Incidence.—As noted before, lime-water bile is an uncommon condition. Phemister, Rewbridge, and Rudisill (1931) found 4 cases in 318 consecutive radiological examinations for gall-bladder disease, but Matthews, quoted in the same paper, found only 1 case in 6700 similar examinations. In the great majority the radio-opaque material is confined to the gall-bladder, and in these the possibilities occur of confusion with calcification of the gall-bladder wall (porcelain gall-bladder) or of being overlooked in the course of routine cholecystography.

Aetiology.—According to McCall and Tuggle (1942) the age and sex distribution of the condition are the same as for other types of gall-bladder disease. The 3 patients described in the present paper, women aged 36, 43, and 57 years, fit in with the usual pattern.

Obstruction of the cystic duct was present in all the reported cases in which the condition was limited to the gall-bladder. Obstruction of the common bile-duct was present in our 3 patients, and in the 3 similar cases in which both the gall-bladder and the common duct were visualized. From this it appears that obstruction at some point in the biliary tract is a pre-requisite of the condition. It is probable that the obstruction occurs initially in the cystic duct in all cases and that the lime-water bile is formed in the obstructed gall-bladder. In a few instances the obstructing stone then moves into the common bile-duct, allowing the opaque bile to flow after it and fill this duct also (Simmonds, 1957). The case reported by Houghton (1952) in which a layer of calcium carbonate was found on the gall-bladder surface of a pigment stone obstructing the cystic duct supports this theory.

Infection of the gall-bladder is usually considered to be necessary for the condition to occur. Its presence is mentioned in most of the published reports of cases. However, we were unable to obtain any growth on culture of swabs taken from the biliary tract at the time of operation in *Case* 2. In *Cases* 1 and 3 bacteriological examination was not performed. In both these patients the gall-bladder showed signs of chronic cholecystitis and it must be assumed that they had been infected at some time. The role of infection in the production of gall-stones has been shown by experiments such as those of Wilkie (1928) and Phemister, Day, and Hastings (1933). In these experiments infection of the gall-bladders of rabbits with patent cystic ducts resulted in the formation of cholesterol stones, whilst both in rabbits and dogs such infection after the cystic duct had been ligated produced calcium carbonate stones.

The combination of gall-bladder infection and cystic duct obstruction occurs in many cases of gall-bladder disease without the formation of lime-water bile. An additional factor appears to be necessary to produce this condition. It does not appear to be an abnormality of calcium metabolism, because abnormal serum-levels of calcium and phosphorus have not been found in any of the reported cases except that of Schubb and Goodstone (1933). Various theories have been suggested to account for this extra factor. McCall and Tuggle (1942) postulated that inflammation in the gall-bladder produced a fall in the cholesterol and bile-salt content of the bile and that this led to the precipitation of calcium salts. Drury, McMaster, and Rous (1924) showed that the reaction of the bile excreted by the liver is distinctly alkaline, the average *p*H being 8·2, whereas that of the bile in the gall-bladder is acid, with a normal range of 5·2–5·6. This change appears to take place in the gall-bladder. It may fail to occur in the presence of infection or inflammation so that the hydrogen-ion concentration of the gall-bladder bile then remains the same as that of the liver bile. This is of importance with regard to the type of calcium salt which forms. In nearly every case of lime-water bile in which the salt was analysed it was found to be pure calcium carbonate. A few cases were calcium bilirubinate, and in these the substance in the biliary tract was black in colour, whilst some others had a mixture of carbonate and bilirubinate. Calcium carbonate will only crystallize from solution when the *p*H is greater than 6·6 and its formation should not occur in the normal gall-bladder bile (normal range 5·2–5·6).

Other workers have attempted to explain the formation of the calcium salts not on the basis of increased precipitability of calcium but on the theory that the amount of calcium in the gall-bladder may be increased. Schubb and Goodstone (1933) believed that inflammation of the gall-bladder increased the concentration of calcium in the contained bile and thus predisposed to its precipitation. Although they quoted similar opinions from various sources to support their theory, they did not produce any evidence to show that a rise in the calcium content actually occurs under such circumstances. It is interesting to note that their case was the only one

reported in which hypercalcæmia was found. Their theory is supported, however, by the findings of Johnston, Ravdin, Austin, and Morrison (1932), that in dogs under experimental conditions calcium is not secreted into the normal gall-bladder but may be secreted in small amounts if the gall-bladder is damaged.

The rapid formation of lime-water bile (Baron, 1940) may be explained by the process described by Fowweather and Collinson (1927). When rapid crystallization occurs in a supersaturated solution, pure crystals of the salt are formed and these will tend to remain in suspension. On the other hand, if a preformed nucleus is present, slow crystallization will take place around it as soon as the concentration of the solute exceeds the saturation value; and this process will continue slowly until a stone forms. As the concentrations of the various salts present in physiological fluids vary from time to time, such a stone will contain a mixture of crystals of the different salts present in the solution. The findings of a pure suspension of calcium carbonate in the majority of the reported cases suggests that rapid crystallization from supersaturated solution has taken place and this fits in with Baron's observations that the condition can appear over the course of a few days.

Symptoms and Signs of Lime-water Bile.—There do not appear to be any clinical symptoms or signs by which the condition can be diagnosed. Every reported case has had pain varying from a slight ache to severe colic. The duration of symptoms has varied from one week to over ten years. Jaundice is a variable feature. McCall and Tuggle (1942) found no jaundice in the 15 cases they collected, but in all of these the obstruction was in the cystic duct. In the 6 cases with common-duct obstruction (Cohen, 1955; Simmonds, 1957; present authors) jaundice was present. Pancreatitis was found at operation in 2 cases (Simmonds, 1957; *Case* 2 in the present review), although in neither had the diagnosis been suspected before operation.

Radiological Appearances.—These are of great interest. In the 3 cases recorded here the radio-opacity of the material was such that the shape and outline of the common duct could be appreciated and consequently the diagnosis was not difficult. The limy material in the gall-bladder was seen in *Case* 2 to form a horizontal layer in the erect position (*see Fig.* 225). The stone impacted at the lower end of the duct was clearly seen in all cases and in all oral cholecystography showed a non-functioning gall-bladder.

It is interesting to speculate how frequently lowered concentration of limy bile occurs without casting a sufficiently dense radiographic shadow to allow a positive diagnosis. It is our opinion that less dense shadows are probably frequently overlooked.

In the differential diagnosis other causes of calcification in the right hypochondrium must be considered. The amorphous nature of the calcification and its outline corresponding to the shape of the duct, however, makes the diagnosis relatively easy.

SUMMARY AND CONCLUSIONS

1. Three more cases are added to the literature on lime-water bile.

2. The aetiology, clinical manifestations, and radiological features are discussed.

3. Radiological examination is the only method by which a pre-operative diagnosis can be made.

4. A hitherto unreported radiological finding of visualization of the pancreatic duct by the reflux of limy bile is recorded.

We wish to thank Mr. J. R. Cameron and Mr. Donald McIntosh for permission to publish the cases under their care, and Dr. J. G. Kininmonth, radiologist, for the films of the first case.

REFERENCES

BARON, J. (1940), *Radiology*, **35**, 741.
CHURCHMAN, J. W. (1911), *Johns Hopk. Hosp. Bull.*, **22**, 233.
COHEN, O. H. (1955), *Radiology*, **65**, 78.
DRURY, D. R., MCMASTER, P. D., and ROUS, P. (1924), *J. exp. Med.*, **39**, 403.
FOWWEATHER, F. S., and COLLINSON, S. A. (1927), *Brit. J. Surg.*, **14**, 583.
HOUGHTON, L. W. (1952), *Ibid.*, **39**, 336.
JOHNSTON, C. G., RAVDIN, L. S., AUSTIN, J. H., and MORRISON, J. L. (1932), *Amer. J. Physiol.*, **99**, 648.
KNUTSON, F. (1933), *Acta radiol., Stockh.*, **14**, 53.
MCCALL, M., and TUGGLE, A. (1942), *Amer. J. med. Sci.*, **203**, 413.
PHEMISTER, D. B., DAY, L., and HASTINGS, A. B. (1933), *Ann. Surg.*, **98**, 595.
— — REWBRIDGE, A. G., and RUDISILL, H. (1931), *Ibid.*, **94**, 493.
SCHUBB, T., and GOODSTONE, S. B. (1933), *Surg. Gynec. Obstet.*, **57**, 325.
SIMMONDS, H. T. (1957), *Amer. J. Roentgenol.*, **78**, 1020.
VOLKMANN, J. (1926), *Münch. med. Wschr.*, **73**, 2014.
VOLKMANN, R. (1908), *Ibid.*, **74**, 363.
WILKIE, A. L. (1928), *Brit. J. Surg.*, **15**, 450.

CHRONIC FOLLICULAR CHOLECYSTITIS

RADIOLOGICAL, PATHOLOGICAL AND SURGICAL ASPECTS

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CHRONIC follicular cholecystitis is a type of chronic inflammation of the gall-bladder characterized by the formation of lymph follicles with hyperplastic germinal centres in all layers of the gall-bladder. In full-blown cases these can be recognized in the gross specimen as numerous prominences of firm consistency, round in shape, and elevated above the inner surface of the gall-bladder. Although frequently encountered in association with typhoid fever, it occurs also in individuals with no detectable antibodies against the *Salmonella* group of organisms. In this latter group the aetiological agent remains uncertain. Anderson's synonym for this condition is 'cholecystitis lymph-follicularis' (1957).

INTRODUCTION

In 1890 Gilbert and Girode recognized that the gall-bladder could be infected during typhoid fever. Chiari (1894) first described inflammatory changes in the gall-bladder mucosa associated with hyperæmia and œdema, and pointed out the high frequency of cultured typhoid bacilli within the gall-bladder during typhoid fever. He obtained pure cultures in 19 out of 22 autopsy cases. Schottmüller and Fraenkel (1925) illustrated their pathological description with photomicrographs showing hyperplastic germinal centres and lymphocytic and plasma-cell infiltration in the mucosa. Later, in 1931, Mallory and Lawson noted in some gall-bladders prominent rugæ and a granular appearance to the mucosa. There was no exudate on the serosa. Although 6 out of 7 cases contained stones, no stones had a cholesterol component. They were the first to observe hyperplastic germinal centres in approximately equal numbers in all portions of the wall of the gall-bladder from the neck to the fundus. They further emphasized that although the lesions were characteristic, they were not pathognomonic of typhoid fever; that the same pathology could be produced by other organisms. They considered the changes within the gall-bladder wall to be the result of persistent infection of the bile itself.

In 1957 we were able to recognize this lesion by radiography in *Case* 2, and the diagnosis of chronic follicular cholecystitis was made, after removal of the gall-bladder, by the pathologist.

CASE REPORTS

Case 1 (Service of the late Dr. A. T. Bazin).—Miss L. S., born in 1893, had in 1922, at the Sherbrooke Hospital, a cholecystostomy performed for the removal of gall-stones. In 1945, at The Montreal General Hospital, a normal functioning gall-bladder was demonstrated by radiography prior to a gastrectomy for a penetrating duodenal ulcer. The patient contracted typhoid fever, for which she was admitted to the Sherbrooke Hospital on June 27, 1950. She was immediately given chloromycetin 500 mg. every 4 hours. Her temperature, 104·6° F. on admission, became normal on the sixth day, and remained so throughout the rest of her hospital stay. On July 4 the chloromycetin was reduced to 250 mg. four-hourly and discontinued on the evening of July 9. She had no symptoms at any time that would draw attention to the gall-bladder.

After her discharge on Aug. 1, stool cultures were found positive for the typhoid bacillus. At the end of August, 1950, she was given four intravenous injections of typhoid bacteriophage, suggested and supplied by the Quebec Provincial Laboratory. This was an attempt to overcome the carrier state, without success.

She was referred to The Montreal General Hospital for cholecystectomy in another attempt to overcome the carrier state. When admitted on Feb. 7, 1951, the patient had regained her usual weight and had no residual gastro-intestinal symptoms.

On examination she was a well-developed, well-nourished white female in no distress. B.P. 145/90. Pulse 78, regular. Abdomen was slightly rounded and obese. No masses or organs were palpable. A poorly defined area of slight tenderness was present in the right upper quadrant. There was no rebound tenderness.

Prior to the operation the urinalysis was negative but the stool cultures showed a heavy growth of *S. typhosa*. Cholecystography failed to demonstrate any evidence of a functioning gall-bladder or radio-opaque calculi.

On Feb. 16 a cholecystectomy was performed. The gall-bladder was submitted in a sterile container for confirmation of the typhoid bacterial infection. Cultures were made of the bile and *S. typhosa* was cultivated. The organ measured 11 × 4 × 3 cm. and had a smooth glistening serosa. It was distended by a rather mucoid bile content and three small cholesterol calculi were impacted within the spiral valve of the neck. The wall was of normal thickness. The mucosa was thick, velvety, and brown in colour.

The microscopic sections from the fundus, the body, and the neck exhibited no fibrosis, but a mild mural thickening due to muscular hypertrophy and to inflammation in the mucosa. A few Rokitansky-Aschoff sinuses penetrated the muscle coat. Throughout the mucosa there was an intense, diffused infiltration by lymphocytes and plasma cells and very occasional polymorphonuclear leucocytes. In addition there was well-marked lymphoid follicle formation within the lamina propria (*Fig.* 228). There were small foci of chronic inflammatory cells within the muscle coat. The pathological diagnosis given was heavy typhoid infection of the gall-bladder with cholelithiasis and chronic follicular cholecystitis.

After operation, six successive stool cultures failed to grow *S. typhosa*. The patient was discharged in good condition on March 13, 1951.

Case 2 (Service of Dr. R. L. Estrada).—Miss A. C., born in 1892, had eight admissions to The Montreal General Hospital for unrelated organic conditions prior to 1957. Although she had complained of flatulent dyspepsia for many years, the first attack of colicky right

upper quadrant pain radiating through to the back, associated with anorexia, was in September, 1957. She had no nausea, vomiting, jaundice, fever, or chills during the attack. A second similar episode occurred on Nov. 10, 1957.

Cholecystography was done on Nov. 12, using twelve Telepaque tablets. The examination included the taking of spot films with varying degrees of compression in the upright position. Films were also taken after a fatty meal.

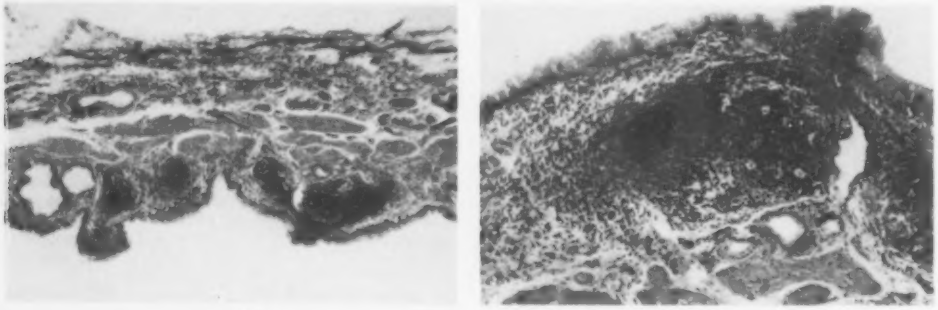
The films revealed a functioning gall-bladder. Within the dye shadow of the gall-bladder there were innumerable small round translucent shadows. These were slightly larger than pin-head in size. They appeared to involve practically the entire gall-bladder (*Fig.* 229). The periphery of the dye shadow showed a rather ragged, irregular appearance. The dye shadow was neither unusually dense nor unusually faint. Response to a fatty meal was normal.

Three possible diagnoses were considered: (*a*) Multiple papillomata; (*b*) Cholesterolosis; (*c*) Translucent gall-stones. Of these three, multiple papillomata appeared to be the most likely diagnosis, the radiographs suggesting the presence of numerous small discrete masses, either mucosal or submucosal, or both.

Admission to hospital was advised in view of radiological findings compatible with papillomatosis of the gall-bladder.

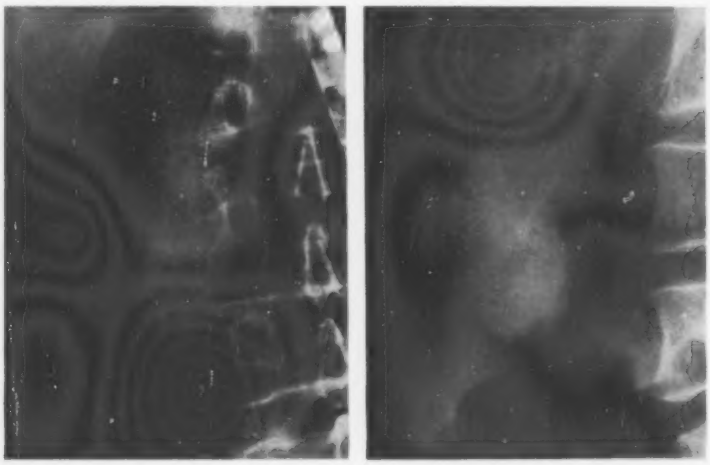
On Nov. 25 physical examination revealed a well-developed and well-nourished white female, alert, co-operative, and in no distress. B.P. 174/80. Pulse 76, regular. Respirations 20. The abdomen was soft and flat, with a healed right lower quadrant scar. Murphy's sign was positive. No masses were palpable. Bowel-sounds were normal. There was no anæmia. The white and differential counts were normal.

On Nov. 28 a cholecystectomy was performed. The enlarged, lobulated, cholecystic lymph-node present was removed with the specimen. The gall-bladder was 7·5 cm. in length by 5 cm. in circumference. In the fresh state the serosa was smooth, glistening, and slightly congested. On opening it the mucosa had a reddish appearance (*Fig.* 230 A). It was studded with numerous prominences, 3–5 mm. in diameter and raised up to 3 mm. above the otherwise smooth mucosa. Approximately one-third of the surface of the gall-bladder was covered by these prominences.



A B

FIG. 228—*Case* 1. A, Intense and diffuse infiltration throughout the mucosa by lymphocytes and plasma cells forming lymph follicles in the lamina propria. Note the foci of chronic inflammatory cells within the muscle coat. The epithelium is intact. (× 12.) B, Lymph follicle in lamina propria. (× 36.)



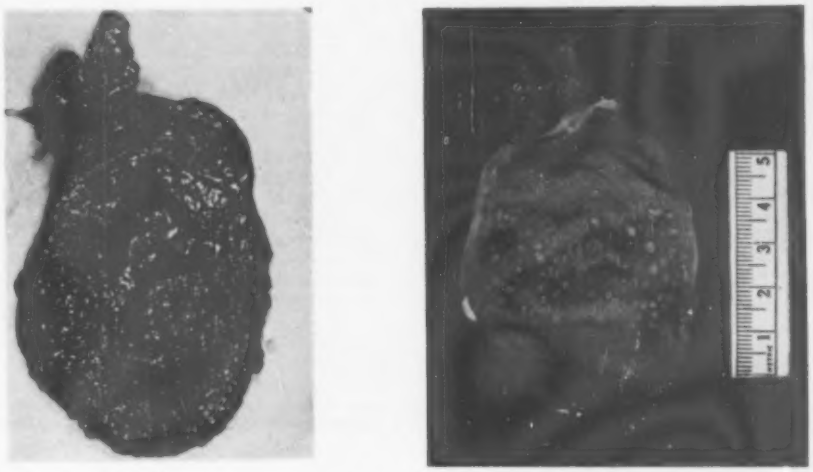
A B

FIG. 229.—*Case* 2. A, Cholecystogram following the ingestion of twelve Telepaque tablets. Note the faint dye shadow and the numerous round translucencies causing a ragged and irregular outline of the dye shadow. B, The general appearance does not change with altered position of patient.

After fixation in formalin, the serosa, slightly congested, was smooth and shiny. The mucosa was green-yellow in colour, had a velvety appearance, but was studded with numerous firm, rounded, elevated nodulations, 1–3 mm. in size, and covering about one-third of the mucosal surface (*Fig.* 230 B). There were no calculi present.

The microscopic sections showed multiple mucosal excrescences or pseudopolypoid protrusions due to the formation within the mucosa of well-formed lymphoid follicles, abutting on the muscular coat. There were fewer penetration by Rokitansky-Aschoff sinuses. The cholecystic lymph-node revealed prominent germinal centres in well-developed lymphoid follicles and sinusoidal phagocytosis of a brown pigment. The pathological diagnosis was chronic follicular cholecystitis.

The bile was sterile. The fæcal flora was normal. The Widal test was negative for all antigens. The patient since discharge from hospital takes dehydrocholic acid for three days monthly and must follow a low-fat diet, otherwise she gets severe epigastric pain similar to the pain she



A B

FIG. 230.—*Case* 2. A, Appearance of the gall-bladder mucosa in the freshly opened specimen showing the prominent lymph follicles raised above the otherwise smooth mucosa. B, After fixation in formalin the mucosa has taken a greenish-yellow colour. The prominences are slightly smaller and although they simulate cholesterol stones they are actually hyperplastic lymph follicles covered by intact mucosa.

A B

FIG. 231.—*Case* 2. A, Intramucosal lymphoid follicle forming a pseudopolyp within the gall-bladder. (× 60.) B, Lymph follicle formation deep to muscularis in gall-bladder wall. (× 25.)

follicles present in the perimuscular fibrous tissue of the outer coat (*Fig.* 231). Lymphocytic and other inflammatory-cell infiltration was very meagre. Moderate hypertrophy of the muscularis was associated with a had prior to the operation. Intravenous cholangiography in July, 1959, demonstrated that the common bile-duct was dilated as compared with the normal. Since the duct was normal in calibre at the time of surgery, this would

suggest obstruction in the region of the ampulla of Vater. This must be incomplete, as contrast media are noted in the duodenum. Barium series demonstrated the duodenal loop to be normal.

DISCUSSION

Our experience is that chronic follicular cholecystitis is much less frequent than the reported 6·5 per cent incidence (Mallory and Lawson, 1931).

In a review of 2473 gall-bladders removed surgically over a ten-year period (1949–1958 inclusive) at The Montreal General Hospital, only 2 gall-bladders could be so diagnosed, an incidence of 0·08 per cent. In 1957 there were 347 cholecystectomies performed and the disease observed was as follows:—

Acute cholecystitis	26 cases (7·5 per cent)
Acute and chronic cholecystitis	101 cases (29·1 per cent)
Chronic cholecystitis	218 cases (62·6 per cent)
Tumours—benign*	5 cases (1·5 per cent)
Tumours—malignant	1 case (0·3 per cent)
Biliary calculi present in	302 cases (87·0 per cent)
Associated appendectomy performed in	104 cases (29·9 per cent)

* Four of the five gall-bladders in this group had chronic cholecystitis associated with benign tumor.

The 218 cases of chronic cholecystitis were then subdivided according to their microscopic appearance in the following manner:—

Chronic inflammatory-cell infiltration	181 cases (83·1 per cent)
Chronic inflammatory-cell infiltration with occasional poorly developed microscopic lymph follicles widely scattered in all coats (all cases associated with cholelithiasis)	12 cases (5·5 per cent)
Chronic follicular cholecystitis with macroscopic hyperplastic lymph germinal centres in all gall-bladder coats (10 cholelithiasis)	1 case (0·4 per cent)
Cholesterolosis	24 cases (11·0 per cent)

We would like to emphasize that the term 'chronic follicular cholecystitis' or 'cholecystitis lymphfol .cularis' does not apply to the poorly developed microscopic lymph follicles seen in about 5 per cent of chronic cholecystitis. The term only applies to the chronic gall-bladder inflammation with macroscopic lymph follicles and hyperplastic germinal centres within all coats of the gall-bladder.

The diagnosis of chronic follicular cholecystitis prior to any operative interference is a difficult one. In the presence of, or with a history of, typhoid fever, one can suspect this condition. It has been demonstrated that the incidence is greater amongst patients with typhoid fever (even though they have no symptoms referable to the gall-bladder) and that *S. typhosa, per se*, does not cause chronic follicular cholecystitis. Although persistent infected bile has been incriminated, we have observed chronic follicular cholecystitis in the presence of sterile bile. It is, however, possible that bile could have been infected in the past, but we have no evidence to substantiate such a premise. Whether a virus infection could cause this type of pathological change is not known. Although the possibility exists, our patients have not had the leucopenia of virus infection when under our care. In the absence of typhoid fever and of infected bile, the diagnosis is much more difficult. The symptomatology is that of chronic cholecystitis, with or without biliary calculi.

As an aid in diagnosis cholecystography can be done. Our above-mentioned experience (*see above*) led us to consider three possible diagnoses: (*a*) Multiple papillomata; (*b*) Cholesterolosis; and (*c*) Translucent stones. Of these, papillomatosis appeared to be the most likely diagnosis.

In considering cholesterolosis in the differential diagnosis, one must remember that in this condition there is often an *unusually dense* dye shadow. Gall-bladder mucosa has normal functioning capacity, or even better than normal capacity. Furthermore, in cholesterolosis, the gall-bladder is unusually contractile in response to a fatty meal. This was not the case in our patient. The masses of cholesterol which adhere to the mucosa cause translucent shadows in the radiographs. However, these shadows are usually irregular in outline. They do not change in position regardless of the position of the patient. This is the only respect in which the findings in our patient resemble those of cholesterolosis.

The possibility of cholelithiasis was not seriously considered. Small translucent stones can usually be seen to move with varying positions of the patient, and with varying degrees of compression. Also, one never sees a ragged or irregular outline of the periphery of the dye shadow in the presence of gall-stones.

After consideration of all those factors it appeared that the gall-bladder was extensively involved by small papillomata, lying immediately beneath the mucosa and projecting out into the lumen. Despite this extensive involvement by disease, the mucosa still retained to a considerable degree its ability to function.

When the histological diagnosis of chronic follicular cholecystitis was reported in our patient, a search of the radiological literature was made for similar cases. Standard text-books (Schinz, Baensch, Friedl, and Uehlinger, 1954; Shanks and Kerley, 1952), one of which has an extensive bibliography on the gall-bladder (Schinz and others) (about three hundred and fifty references in all), made no reference in this condition in their text or in the bibliography. The *Index Medicus*, reviewed from 1938 to 1958, also failed to yield any radiological reference to a publication on this subject. Jones and Walker (1957) did not observe it among the 48 tumours and pseudo-tumours of the gall-bladder they reported. Consequently, we infer that chronic follicular cholecystitis is an extremely rare condition.

Are there, therefore, any specific radiological criteria which will enable us to recognize and identify this very rare condition in the future? We believe that there are, provided the gall-bladder is functioning. Baz and Perrin (1953) and Perrin (1953) have reported non-function of the gall-bladder. In their cases, as in our *Case* 1, non-function of the gall-bladder was due to blockage of the cystic duct by cholesterol calculi. In the absence of biliary calculi the key points in the diagnosis of chronic follicular cholecystitis appear to be a gall-bladder showing a faint dye shadow because of impaired function; numerous small round translucencies casting a ragged, irregular outline of the dye shadow as demonstrated in the cholecystogram; and the fact that this appearance does not change as the patient is positioned. Probably the disease could be so far advanced as to cause non-function, either by blockage by the giant

lymph-follicles at the origin of the cystic duct or by compression of an enlarged cystic lymph-node, but we have no precedent for assuming that this could occur.

This rare condition is recognizable radiologically if the above-mentioned considerations are kept in mind. The diagnosis of chronic follicular cholecystitis should sometimes be included in the differential diagnostic possibilities, even though it is of such rare occurrence.

The treatment of symptomatic chronic cholecystitis with lithiasis is cholecystectomy, and also probably the same applies to the case with silent gall-stones. There are two conditions which we consider lend themselves to cholecystectomy in the non-calculous gall-bladder, viz.: (1) The typhoid carrier; and (2) The individual with either single or multiple tumours of the gall-bladder without stones, diagnosed in the course of investigation by cholecystography (Jones and Walker, 1957; Kirklin, 1931).

The typhoid carrier has had the organisms in the bile during the attack of typhoid fever. He may have had gall-stones, either of calcium or cholesterol, prior to the attack of typhoid fever, or he may develop gall-stones years later, from which the typhoid bacillus may be cultured. Even if stones do not develop, the bacilli can continue to grow in the bile—and are excreted in the stool. This becomes a hazard to the community. Cholecystectomy and choledochostomy have been recommended in these cases (Aird, 1949). The T-tube in the common bile-duct is removed when the bacillus is no longer cultured. According to Aird cholecystectomy is no guarantee that infectivity will cease.

The papilloma, either single or multiple, is liable to undergo malignant changes, or may be malignant from the beginning. For this reason cholecystectomy should be recommended. The fact that the patient may have chronic follicular cholecystitis should not change one's advice. It is far better to remove this inflammatory condition than to allow a malignancy to develop or progress.

During operation one has encountered numerous avascular peritoneal adhesions between the gall-bladder and adjacent structures. The foramen of Winslow has been patent. The gall-bladder has been compressible if there were no biliary calculi present. Even with the bile expressed, careful palpation of the wall prior to removal failed to convince one that there was a palpable intramural tumour present. A clue as to the possible diagnosis of chronic follicular cholecystitis is the cystic lymph-node which is enlarged (approximately $1{\cdot}5 \times 1$ cm.), partly lobulated, and of quite firm consistency. Once the gall-bladder has been removed, the gross and microscopic appearance is as we (*see above*) and others (Anderson, 1957; Baz and Perrin, 1953; Fauvert, 1948; Mallory and Lawson, 1931; Perrin, 1953; and Schottmüller and Fraenkel, 1925) have described.

SUMMARY

1. Chronic follicular cholecystitis, or cholecystitis lymph-follicularis, has been defined.

2. Two cases have been reported, one with *S. typhosa* infection and one in which the aetiological agent remains unknown.

3. This condition can be diagnosed radiologically. Its characteristic features and differential diagnosis have been outlined.

4. The incidence, surgical problems, and features have been detailed.

5. The surgical, the gross, and the microscopic pathology have been illustrated and described in detail.

6. Despite the previously held belief that cholesterol stones were never found with chronic follicular cholecystitis, it is our experience, and that of others (Baz and Perrin, 1953; Perrin, 1953), that such is not the case.

7. Chronic follicular cholecystitis has been found to occur in the absence of detectable bile infection and in the absence of biliary calculi.

We wish to thank Dr. W. H. Mathews and Dr. H. Rocke Robertson, of The Montreal General Hospital, for their kind advice and criticism in the preparation of this manuscript.

REFERENCES

AIRD, IAN (1949), *A Companion in Surgical Studies*, 807–808. Edinburgh: E. & S. Livingstone.

ANDERSON, W. A. D. (1957), *Pathology*, 3rd ed., 826. St. Louis: C. V. Mosby Co.

BAZ, G. G., and PERRIN, T. G. (1953), *Gac. méd. Méx.*, **83**, 453.

CHIARI, H. (1894), *Z. Heilk., Berl.*, **15**, 199.

FAUVERT, RENÉ (1948), *Paris méd.*, **135**, 344.

GILBERT, A., and GIRODE, J. (1890), *Sem. méd. (Paris)*, **10**, 481.

JONES, H. W., and WALKER, J. H. (1957), *Surg. Gynec. Obstet.*, **105**, 599.

KIRKLIN, B. R. (1931), *Amer. J. Roentgenol.*, **25**, 46.

MALLORY, T. B., and LAWSON, G. M. (1931), *Amer. J. Path.*, **7**, 71.

PERRIN, T. G. (1953), *Rev. clin. españ.*, **51**, 305.

SCHINZ, H. R., BAENSCH, W. E., FRIEDL, E., and UEHLINGER, E. (1954), *Roentgen Diagnostics*, vol. 4, 3715–3723. New York: Grune & Stratton.

SCHOTTMÜLLER, H., and FRAENKEL, E. (1925), *Münch. med. Wschr.*, **72**, 2150.

SHANKS, S. C., and KERLEY, P. (1952), *A Text-book of X-ray Diagnosis*, 2nd ed., vol. 3, 461–487. London: H. K. Lewis.

INCARCERATED INGUINAL HERNIA IN INFANCY WITH TESTICULAR GANGRENE

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INCARCERATION of small bowel in an inguinal hernia in infancy is a common occurrence. A very rare complication of this condition is an associated testicular gangrene due to vascular compression, and 2 such cases were described by Bennett-Jones (1937).

FIG. 232.—Anteroposterior erect radiograph of the abdomen showing fluid levels and increase in intraperitoneal free fluid.

FIG. 233.—Lateral erect radiograph. Distension of the abdomen is well shown.

In 45 cases of incarcerated or strangulated infantile herniæ collected by Rendle-Short and Havard (1954) none had gangrene of the testicle. Thorndike and Ferguson (1938) studied 1740 herniorrhaphies in children and found 'strangulation' in 106, of which 45 occurred in the first year of life, but 'true strangulation' was only present in 5 cases and none suffered the complication of gangrene of the testicle.

CASE REPORT

J. T., a 6-week twin male infant, 1 month premature and weighing 5 lb. 6 oz., was admitted on Aug. 15, 1959, from another hospital. His mother had noticed a swelling in the right scrotum 40 hours before admission, when the infant refused his feeds and whimpered with pain. Later he vomited bile-stained fluid and the stools became scanty. During the next 24 hours he continued vomiting, the vomitus becoming brown and offensive, and he passed small amounts of brown mucus per rectum. The mother noted that the scrotal swelling was now larger, red, and hot. His admission was arranged.

ON EXAMINATION.—He was a dehydrated, ill-looking infant with a pyrexia of 101° F. The abdomen was distended and showed visible coils of small bowel. The right scrotum was red and œdematous and contained a firm, tender, irreducible mass. No bowel-sounds were heard on auscultation, and the rectum was empty. A radiograph of the abdomen showed dilated loops of small intestine with fluid levels, consistent with small-gut obstruction. There was also an increase in intraperitoneal free fluid (*Figs.* 232, 233). The pre-operative diagnosis was strangulation of small bowel in an inguinal hernia with peritonitis. The infant was resuscitated for 4 hours with intravenous therapy and gastric aspiration.

AT OPERATION.—The abdomen was opened through a right transverse rectus-cutting incision just below the umbilicus. The small bowel was distended and there was turbid free fluid in the peritoneal cavity. An obstructed loop of ileum was found entering the right inguinal canal. A transverse incision was then made in the inguinal region and a distended and œdematous cord containing a gangrenous testis delivered into the wound. After manipulation of the cord the incarcerated bowel was found to have reduced itself and was perfectly viable, the only evidence of incarceration being a shagginess of the peritoneal coat of the terminal ileum. Orchidectomy was performed with division of the cord at the internal ring. Both incisions were closed without drainage.

Intravenous therapy was continued for 48 hours, by which time the baby was having normal stools. Penicillin and streptomycin were given for 8 days after operation. On the thirteenth post-operative day 130 ml. of blood was administered. When discharged from hospital his condition was satsifactory and he was feeding well and gaining weight.

THE SPECIMEN.—Macroscopically the testicle was gangrenous and measured 0·8 cm. in diameter. The gangrene was limited to the testicle, the epididymis being congested and the cord normal apart from œdematous changes. There was no evidence of torsion of the spermatic cord (*Fig.* 234).

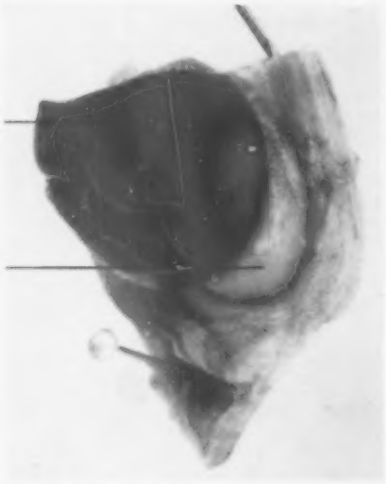

FIG. 234.—Operative specimen showing gangrene of the testicle with a congested epididymis. The irregular incision in the tunica albuginea was made at operation with the organ in situ. (× 3.)

Histologically the testicle showed diffuse interstitial hæmorrhage and gangrene with loss of structure. The epididymis also demonstrated hæmorrhage into its substance, but was not gangrenous. The tunica vaginalis had a fibrinous exudate on its surface. The vas deferens was normal and the spermatic vessels showed no evidence of thrombosis or embolism. The macroscopical and histological appearances were consistent with a vascular obstruction to the testicular vessels.

Culture of the swab taken from the peritoneal cavity grew coagulase-positive staphylococci sensitive to penicillin, streptomycin, and chloromycetin. A later culture from the infected incisions grew coagulase-negative staphylococci.

DISCUSSION

Bennett-Jones (1937) stated that a strangulated or impacted hernia can cause at first a passive congestion and then strangulation of the testicle, and this appears to be the aetiology in this case. Another unlikely aetiology is that the condition was primarily an acute orchitis with ascending funiculitis and adhesion of small bowel to the cord at the internal ring. No history of neonatal umbilical sepsis or orchitis was elicited so this explanation is improbable in this case, although it would better explain the staphylococcal peritonitis and wound infection. The small bowel need not necessarily be strangulated or tightly impacted in the inguinal hernia to produce gangrene of the testicle. Five fatal cases were reported by Rendle-Short and Havard (1954) and, of these, 2 cases only had strangulation with gangrene of bowel, the other 3 cases being merely incarcerated. In the present case the bowel was easily released from the hernial sac by manipulation of the cord and, apart from evidence of adhesion, the bowel itself was perfectly viable.

SUMMARY

A case of impacted inguinal hernia in infancy with testicular gangrene is described and the aetiology discussed.

I would like to acknowledge my thanks to Mr. D. J. Waterston for permission to publish this case, and to Dr. M. Bodian for the pathological report.

REFERENCES

BENNETT-JONES, M. T. (1937), *L'pool med.-chir. J.*, **45**, 121.
RENDLE-SHORT, J., and HAVARD, C. (1954), *Brit. med. J.*, **1**, 680.
THORNDIKE, A., and FERGUSON, C. F. (1938), *Amer. J. Surg.*, **39**, 429.

EXPERIMENTAL SURGERY

ENDOTHELIAL GROWTH IN NYLON VASCULAR GRAFTS

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Since the introduction of prosthetic vascular grafts there have been many reports in the literature of the properties of the various types of materials and of their macroscopical and microscopical appearances after varying periods in use as a vascular conduit. It is known that the graft material becomes incorporated in layers of fibrous tissue covering both its internal and external surfaces, and infiltrating through the framework of the mesh itself. It has also been noted that a smooth shiny layer appears on the luminal surface of the graft (*Fig.* 235). This layer has the appearance of being continuous with, and similar in nature to, the intimal surface of the host vessel, and has been variously described as 'endothelium' (Poth, Johnson, and Childers, 1955; Griffith, Eade, Zech, and Harkins, 1955; Shumway, Gliedman, and Lewis, 1955; Pierpont and Blades, 1957), 'elongated cells apparently endothelium' (Martinez, Dahl, and Grindlay, 1957), 'flattened fibrous tissue' (Harrison, 1957), 'healing' (Harrison,

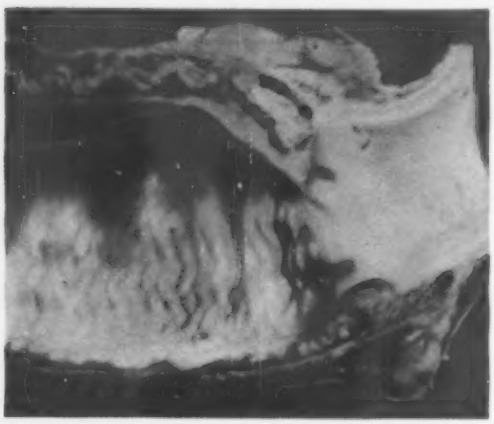

Fig. 235.—Graft after removal with host vessel on the right. The shining film can be seen spreading across the anastomosis and graft.

Fig. 236.—Section of the organized fibrin at right angles to the luminal surface showing numerous superficial cell nuclei, the innermost of which protrude into the lumen.

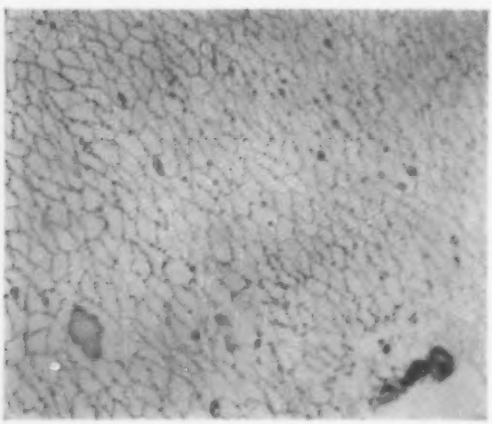

Fig. 237.—Endothelial cell outlines seen *en face* after stripping from graft. 111 days.

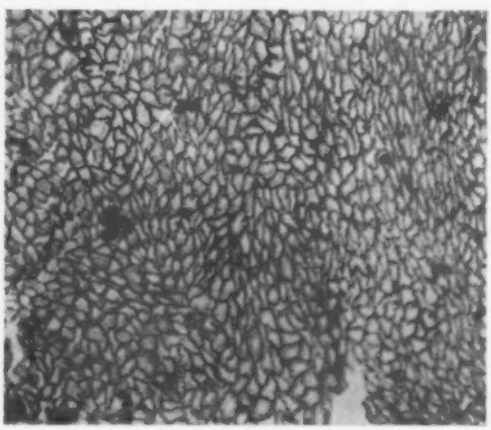

Fig. 238.—Endothelial cell outlines. 209 days.

* Supported by a grant from the Life Insurance Medical Research Fund of Australia and New Zealand.

1959), 'innermost layer indistinguishable from intima' (Kinmonth, Taylor, and Lee, 1955), 'flat endothelial-like cells' (Shumacker, Kajikuri, Grice, Rodriquez, Riberi, Moore, and Siderys, 1957), 'fibrous tissue serving as endothelium' (Sanger, Taylor, McCall, Duchesne, and Lepage, 1956),

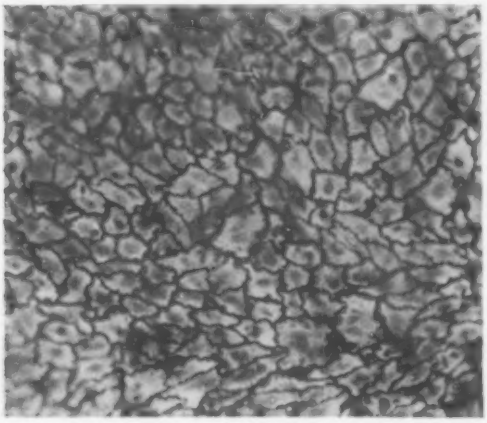

FIG. 239.—Endothelial cell outlines and nuclei. The cell outlines are irregular in size and shape and some large multinucleated cells can be seen. 140 days.

'neo-intima' (Bain, 1957), 'pseudointima' (Szilagyi, Whitcomb, and Claibourne, 1957).

These descriptions have been based on the appearance as seen macroscopically, or in transverse or longitudinal sections (Benjamin, Turbak, Hannon, Shumway, and Lewis, 1957). In the former case there is confusion in the differentiation between the terms 'endothelium' and 'intima', and in the latter cases there is inaccuracy as a series of nuclei only can be seen protruding into the lumen (*Fig.* 236), and, beneath this, layers of fibrous tissue in stages of maturation. The true nature of the innermost lining cell cannot be judged from this type of section but must be seen *en face* (Petry and Heberer, 1957).

The objects of this work were: (1) To determine the identity of this innermost lining by horizontal sheet and stretched preparations in which this layer is removed separately from the rest of the graft lining; (2) To ascertain its origin and speed of formation; and (3) To correlate these with the fibrous invasion of the graft material.

MATERIAL AND METHOD

Thirty adult greyhound dogs were used in the experiments. Crimped nylon prostheses (Edwards-Tapp), 6 mm. diameter and 2–8 cm. in length, were implanted into the femoral artery in one of the following ways: (i) short segments by end-to-end anastomosis; (ii) short by-pass grafts by end-to-side anastomosis; (iii) long by-pass grafts from the iliac to the femoral artery. The grafts together with 1·5 cm. of the host vessel at each end were removed after varying intervals of from 19 to 206 days. After removal the specimens were opened longitudinally, pinned out and stretched, and stained and fixed as suggested by McGovern (1955). The innermost layer was then removed by careful dissection from the centre of the graft and from either end near the anastomotic lines. The specimens were then mounted, studied microscopically, and photographed. The dissection was difficult initially, but with practice became regularly successful although the specimens obtained tended to be fragmentary, but there was always sufficient material to show good histological detail.

In addition to the sheet preparations transverse sections through the graft were prepared.

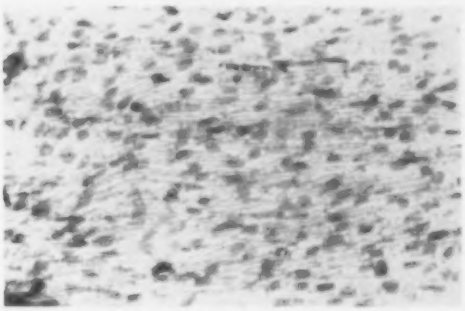

FIG. 240.—Cell outlines and nuclei near suture line. The cell outlines are more regular in size and shape and appear to contain one nucleus only.

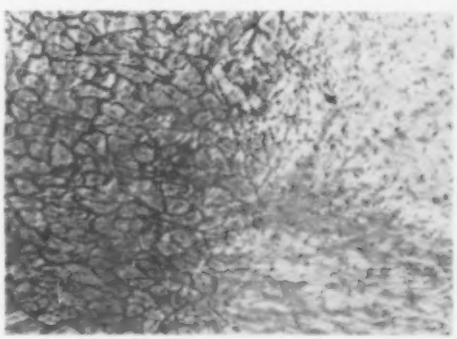

FIG. 241.—Endothelial cells on the left and the sub-endothelial fibrous tissue on the right.

A composite picture of the changes in the wall and lumen of these nylon prostheses was then built up from these two sets of preparations (Deterling and Bhonslay, 1955).

RESULTS

Of the thirty experimental animals used, 3 died directly as a result of the operative procedure and 7 died of causes unrelated to the experiments. In the surviving animals 16 grafts were patent and 4 thrombosed at the time of removal.

The thin fragile innermost layer showed the characteristics seen in *Figs.* 237, 238. The silver stained mosaic pattern of the cell outlines in *Figs.* 237, 238 and the cells and their nuclei in *Figs.* 239, 240 conform to the histological details of endothelial cells. Endothelium was recovered in all specimens

from the graft side of the anastomotic line. The presence of endothelium in the centre of the grafts was variable.

In the 19-day graft, 3 cm. in length, endothelium was recovered in a strip stretching 1 mm. from the anastomotic line. In one graft, 3·5 cm. in length, endothelium was recovered from the centre of the graft after 111 days. Endothelium was also recovered from the centre of the 209-day graft, 5 cm. in length.

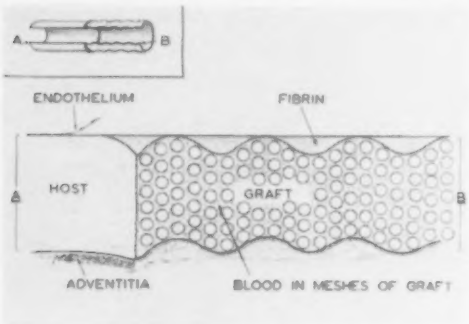

FIG. 242.—Stage 1. The inset shows the host vessel on the left and the graft on the right and the large figure represents the section through the edge AB.

In two grafts, 8 cm. in length and implanted for 63 and 92 days, endothelium was not present in the central zone.

In some of the later experiments we were surprised to find endothelium present in the central region of the graft where the underlying mesh of the graft and the fibrin clot composite could be clearly seen. In earlier experiments some of the specimens were discarded without attempting endothelial recovery from their central zones before it was realized that this could not be judged macroscopically, as will be explained later. Another source of failure was caused by stripping in a plane deep to the endothelium and removing with it the sub-endothelial fibrous-tissue layer (*Fig.* 241).

The transverse sections demonstrated differential rates of organization of the graft material, and of the lining fibrin, in relation to the overgrowing endothelium, and the independence of these two processes.

DISCUSSION

Meijne (1959), in the experimental laboratory of the Surgical University Clinic of the Wilhelmina Hospital, has independently been working on the same subject since 1955 and has recently published the first factual report on endothelial growth in nylon prosthesis. His grafts were inserted into the

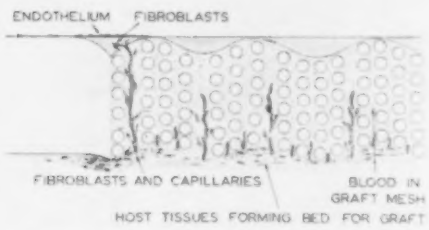

FIG. 243.—Stage 2, with the early growth of granulation tissue through the graft and the spread of endothelium across the first crimp.

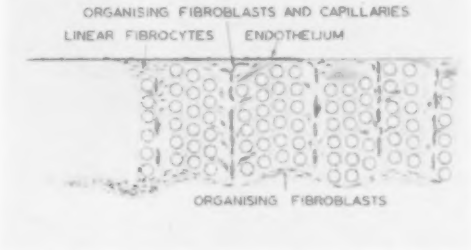

FIG. 244.—Stage 3. Advanced organization in the first crimp, delayed organization farther along the graft. The endothelium is well advanced and the adventitia is well organized.

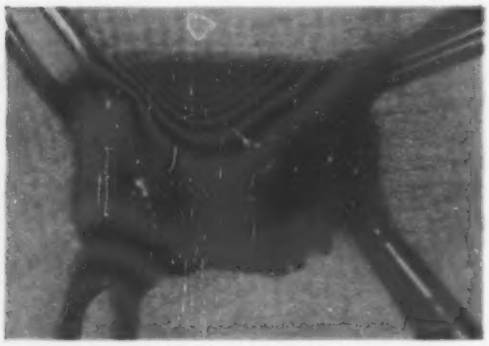

FIG. 245.—The white sub-endothelial fibrous-tissue pannus spreading out from the suture line. Note that this photograph represents only half of the total length of the graft and endothelium was removed from the region of the cut end. 209 days.

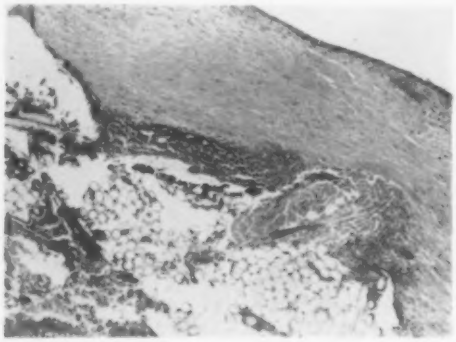

FIG. 246.—The fibrin over the anastomotic line has been well organized and the mature fibrocytes are lying horizontally arranged. The superficial nuclei give the appearance of endothelium. The deeper layers of the fibrin have been replaced by collagen. 209 days.

aorta, abdominal and thoracic, of dogs and goats using non-crimped nylon. He has successfully demonstrated endothelial covering over grafts up to 100 mm. in length. Our findings agree substantially with his.

The mosaic pattern seen in *Figs.* 237–240 has been described by us as that of the cell outline. Florey, Poole, and Meek (1959) have shown by electron microscopy that silver is deposited to some extent on all surfaces of endothelial cells and along cell junctions without exactly delineating either cell membranes or the space between them. For the sake of simplicity we have persisted in our description.

Comparing *Figs.* 239 and 240 it can be seen that the cell outline and nuclei vary considerably in shape and size in these two specimens. In *Fig.* 239 the cell outlines are irregular in size and shape, some being 'giant' in size and multinucleated. In *Fig.* 240 the cell outlines appear to flow longitudinally; the cell outlines are more uniform in size and shape and contain a single nucleus. The difference in cell shape may be due to lack of stretching, to the different elasticity of the implant compared to a normal vessel, especially in the crimps of the nylon, or it may be a manifestation of the immature or growing edge of endothelium, the more elongated and regular cells being more mature and behind the growing edge. Poole, Sanders, and Florey (1958) studied the regeneration of endothelium in the aorta of young rabbits in which the intima of this vessel was mechanically damaged. They found a bizarre pattern of newly formed endothelial cells at the growing edge, many of which were multinucleated. This would appear to confirm our final conclusion in the previous paragraph.

A diagrammatic series of the changes in the graft wall correlated with endothelial overgrowth can be seen in *Figs.* 242–244, 252 and histological confirmation in *Figs.* 246–251.

Stage 1 (*Fig.* 242).—Within a few hours of patent flow the troughs of the crimps of the nylon graft have been filled with fibrin, red blood-cells, leucocytes, and platelets. At the anastomotic lines the internal protrusion of the sutures is also covered. In addition the mesh of the graft is filled with fibrin and cellular elements and there is free blood in varying amounts on the outside of the graft between it and the surrounding tissues.

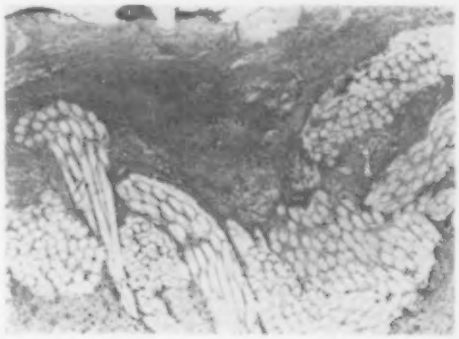

FIG. 247.—The fibrin and cells in the crimp farther along the graft show early invasion only. 209 days.

FIG. 248.—A length of crimped nylon with the anastomosis in the top left-hand corner.

Stage 2 (*Fig.* 243).—Granulation tissue reaction commences in the surrounding tissues. This is found only while the graft remains patent, and varies in degree, depending on the amount of hæmatoma surrounding the graft, and on the nature of the tissues in immediate contact with it. This reaction is brisk when the surrounding hæmatoma is minimal and the graft lies on muscle-sheath or in contact with

serosa or subserosal connective tissue. Similarly if these tissues are closed over the graft, rapid invasion is promoted. The local reaction is slight or delayed if the graft becomes thrombosed or the surrounding

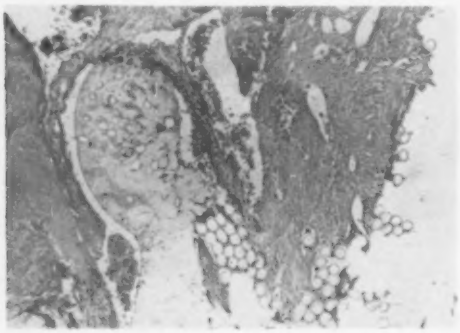

FIG. 249.—Is a large view of the first crimp seen in *Fig.* 248, showing the advanced organization of the fibrin in this crimp and the heat-fused end of the nylon graft.

FIG. 251.—Shows the trough of the first crimp with the organization of the adventitia and the fibrin connected through the mesh of the graft.

hæmatoma is excessive. If one surface of the graft is covered by subcutaneous tissues only, the reaction on this surface is minimal.

This granulation tissue reaction occurs from three main sources:—

a. As described above, from the surrounding tissues.

b. From the ruptured ends of the adventitia of the host vessels.

c. By invasion from the host media following death of the cellular tissue in this zone near the anastomosis. (Richards and Smith, 1958.)

The graft invasion is more rapid therefore in the region of the anastomosis than towards the middle of the graft.

Once the granulation tissue growth has proceeded through the mesh of the graft it begins to invade and organize the fibrin layer.

At the same time endothelium has commenced to grow over the surface of the fibrin. Martinez, Dahl, and Grindlay (1957) have shown that there is a loss of endothelium over the host vessel adjacent

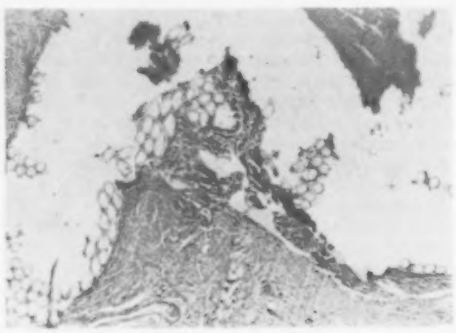

FIG. 250.—Shows the fibrin in the second crimp, as yet not invaded or organized (top right-hand corner.)

to each anastomosis and that this loss is repaired in a few days.

An important point is that the endothelial growth is in advance of the organization of the underlying fibrin and appears to be independent of this process.

The surrounding hæmatoma has been invaded and organized at the same time.

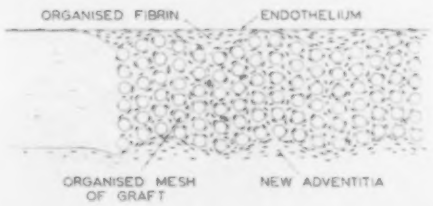

FIG. 252.—Stage 4, with the complete organization of the graft.

Stage 3 (*Fig.* 244).—The fibrin in the first crimp has been invaded and organized and the mature fibrocytes have alined themselves in the direction of flow of the blood-stream. The fibrin in crimps farther along the graft is still as yet not completely organized, but the endothelium has advanced across this region and beyond the areas of complete organization.

The stage of mature organization can be seen macroscopically as a glistening white layer spreading out from the suture line (*Fig.* 245). This is the layer usually described as 'endothelium', etc., as enumerated in the introduction. These descriptions therefore have not distinguished between the endothelium and the sub-endothelial fibrous tissue. Thus Harrison (1959) reported his findings that in a nylon graft 24 months old 'healing' had progressed for only 2–5 mm. across both suture lines—this probably applied to delayed sub-endothelial organization. The white

pannus seen in *Fig.* 245 spreading out from the suture lines is fibrous tissue; endothelium was recovered from the middle section of this graft, although macroscopically it is not apparent and it has obviously spread across this zone independently of sub-endothelial maturation. Microscopical evidence of the differential invasion and organization can be seen in *Figs.* 246, 247. The former shows the organized fibrin layer in the region of the anastomosis, and the latter, early but not complete organization in a crimp farther along the graft. In *Fig.* 248 is seen a length of crimped nylon: the suture line and the heat-fused end of the nylon are in the top left-hand corner. The nylon fibres have been in part dissolved in preparation. The fibrin in the first trough is organized; in the second trough there is early invasion only. This is shown in greater detail in *Figs.* 249–251.

We have recovered endothelium from the mid-section of a 5-cm. graft coated externally with a silicone preparation. On exposure after 186 days the graft was lying free from attachment to the surrounding tissues except at the suture lines. This would appear to be further evidence of the independence of endothelial growth and also evidence of its site of origin.

Stage 4 (*Fig.* 252).—The final stage as seen in this figure shows complete endothelial lining, sub-endothelial fibrous tissue in which the deeper layers are becoming collagenous (Mortensen and Grindlay, 1956), the organized graft, and the new adventitia.

There are very wide variations in the time factors involved in these processes. Under ideal conditions they can be summarized as:—

1. The crimps of the nylon are filled with fibrin within 1–2 hours after release of the clamps.

2. New blood-vessels and fibroblasts appear in the ruptured adventitia by the 5th day, and by the 8th day adventitial and host-tissue granulations have commenced to invade the interstices of the graft in the region of the anastomosis.

3. At the anastomosis the graft has been fully invaded by granulations within 25 days, and by 40 days the invasion of the anastomotic fibrinous inner lining is complete.

4. Farther along the graft invasion is much slower and may take many months, depending on the factors outlined previously.

5. The average rate of growth of endothelium along the graft appears to be of the order of 0·1–0·15 mm. a day from either end.

There are considerable differences in the rate of growth in different grafts. This may be due to technical differences in the degree of host-vessel damage at the time of clamping and anastomosis, or to the difficulty in removing intact this delicate membrane when the underlying tissue is not organized and hence does not form a firm base from which the endothelium may be stripped. If re-endothelialization of these grafts is important in the prevention of thrombosis, these time factors would have a clinical importance.

We believe this endothelial growth is a continuous and contiguous process from the host vessel at either end of the graft (D'Angelo, Benson, and Grimson, 1955). We have not found the endothelial islets described by Voorhees, Jaretzki, and Blakemore (1952), although this could well occur in the sub-endothelial layer. From this work it is unlikely that the endothelium is fibroblastic in origin (Bain, 1957; Wesolowski and Sauvage, 1956) or arises from capillaries near the lumen (Poth, Johnson, and Childers, 1955).

SUMMARY

1. Endothelial growth occurs along crimped nylon vascular prostheses in dogs.

2. This endothelium originates from the host vessel at either end.

3. This growth is independent of the sub-endothelial repair processes.

We are indebted to Mr. S. Woodward Smith and his staff from the Department of Illustration for the reproductions and to Mr. T. C. D. Tedder for his technical assistance in the experiments. Dr. V. J. McGovern and Dr. K. Viner Smith gave valuable assistance in the preparation of the specimens.

REFERENCES

BAIN, WILLIAM H. (1957), *J. R. Coll. Surg. Edinb.*, **3**, 241.
BENJAMIN, ROBERT B., TURBAK, CHARLES E., HANNON, DONALD W., SHUMWAY, NORMAN E., and LEWIS, JOHN, F. (1957), *Ann. Surg.*, **145**, 509.
D'ANGELO, GEORGE, J., BENSON, WALTER R., and GRIMSON, KEITH S. (1955), *Arch. Surg., Chicago*, **70**, 39.
DETERLING, RALPH A., and BHONSLAY, SHIVAJI B. (1955), *Surgery*, **38**, 71.
EDWARDS, W. S. (1957), *Plastic Arterial Grafts*. Springfield, Ill.: Charles C. Thomas.
FLOREY, H. W., POOLE, J. C. F., and MEEK, G. A. (1959), *J. Path. Bact.*, **77**, 625.
GRIFFITH, CHARLES A., EADE, GILBERT G., ZECH, RALPH K., and HARKINS, HENRY N. (1955), *Surg. Gynec. Obstet.*, **101**, 225.
HARRISON, HAROLD J. (1957), *Ibid.*, **104**, 81.
— — (1959), *Ibid.*, **108**, 433.
KINMONTH, J. B., TAYLOR, G. W., and LEE, R. H. (1955), *Brit. med. J.*, **1**, 1406.
MCGOVERN, V. J. (1955), *J. Path. Bact.*, **69**, 283.
MARTINEZ, NESTOR S., DAHL, ELMER V., and GRINDLAY, JOHN H. (1957), *Surgery*, **42**, 1002.
MORTENSEN, J. D., and GRINDLAY, JOHN H. (1956), *Arch. Surg., Chicago*, **72**, 871.
MEIJNE, N. G. (1959), *Arch. Chir. Neerl.*, **11**, 41.
PETRY, G., and HEBERER, G. (1957), *Arch. klin. Chir.*, **286**, 249.
PIERPONT, HOWARD, and BLADES, BRIAN (1957), *Surg. Gynec. Obstet.*, **105**, 370.
POOLE, J. C. F., SANDERS, A. G., and FLOREY, H. W. (1958), *J. Path. Bact.*, **75**, 133.
POTH, EDGAR J., JOHNSON, JOSEPH K., and CHILDERS, JOHN H. (1955), *Ann. Surg.*, **142**, 624.
RICHARDS, H. J., and SMITH, K. VINER (1958), *Med. J. Aust.*, **1**, 450.
SANGER, PAUL W., TAYLOR, FREDERICK H., MCCALL, ROBERT E., DUCHESNE, RONALD, and LEPAGE, GILLES (1956), *J. Amer. med. Ass.*, **160**, 1403.
SHUMACKER, HARRIS B., KAJIKURI, HISAKI, GRICE, PAUL, RODRIQUEZ, RAIMUNDD, RIBERI, ANGELO, MOORE, THOMAS C., and SIDERYS, HARRY (1957), *Surgery*, **41**, 943.
SHUMWAY, N. E., GLIEDMAN, M. L., and LEWIS, F. J. (1955), *Surg. Gynec. Obstet.*, **100**, 703.
SZILAGYI, D. EMERICK, WHITCOMB, JOHN G., and CLAIBOURNE, P. SHONNARD (1957), *Arch. Surg., Chicago*, **74**, 944.
VOORHEES, ARTHUR B., jun., JARETZKI, ALFRED, and BLAKEMORE, ARTHUR (1952), *Ann. Surg.*, **135**, 332.
WESOLOWSKI, SIGMUND A., and SAUVAGE, LESTER R. (1956), *Ann. Surg.*, **143**, 65.

HEPATIC VEIN SPHINCTER MECHANISM IN THE DOG

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SWELLING of the canine liver can be produced by high perfusion pressure, trauma to the organ, anaphylactoid shock, peptone shock, and an injection of histamine. This swelling is thought to be due to obstruction to the outflow of blood from the liver as a result of contraction of hepatic venous sphincters. Such sphincters are known to be present in several animal species, including the dog, and there is some evidence of their presence also in man (Gibson, 1958, 1959). However, the exact site of the sphincter mechanism is still in doubt. Three possible sites have been described. First, at the junction of the hepatic vein with the inferior vena cava (Bauer, Dale, Poulsson, and Richards, 1932); secondly, at the sublobular vein level (sluice valve of Deysach, 1941); and thirdly, at the sinusoidal level (Knisely, Harding, and Debacker, 1957). Alternatively, the whole hepatic venous system may react by vasoconstriction (Maegraith, Andrews, and Wenyon, 1949; Thomas and Essex, 1949).

FIG. 253.—Injection of thorotrast into the left hepatic vein in a dog. There is a block in its passage into the inferior vena cava with resulting backflow into a lobe.

Our own interest in the site of the hepatic sphincter arose from an observation made during injection of the hepatic vein of a dog with radio-opaque dye (*Fig.* 253). The development of a method for cannulation of the left hepatic vein in dogs (Shoemaker, Walker, Van Itallie, and Moore, 1959) led us to study the possible site of this sphincter by means of cinéradiography using an image intensifier.

MATERIAL AND METHODS

The left hepatic vein in the dog may be approached through a midline upper abdominal incision and separation of the left lateral and left central lobe. In the fissure between these lobes lie the hepatic vein, a branch of the portal vein, and bile-duct. A purse-string suture of 00000 silk on an atraumatic needle is inserted through the coats of the vein, an incision is made in the centre of this, and a cannula is inserted for about 1·0 cm. The cannula is fixed by the silk suture and is led out through a stab incision in the flank. It is flushed through with normal saline and a spigot is inserted. Michel clips are placed at the site of the vein, on the inferior vena caval opening in the diaphragm, and near the inferior vena cava below the liver. These served to mark movements of the diaphragm in relation to phases of respiration.

This procedure was carried out in 5 dogs who were then allowed to recover from anæsthesia and about 4–8 hours later were given a further injection of pentobarbitone before the radiological examination. This consisted in the injection down the hepatic vein cannula of 50 per cent diodone, the flow being recorded by means of a Philips image intensifier with attached camera working at a speed of 25 frames per second.

Injection of Histamine.—Control films were taken of each animal and histamine acid phosphate in varying doses was administered down the hepatic vein cannula followed by 10–15 ml. of diodone.

Trauma to the Liver.—This was tested in one animal. The hepatic vein cannula was inserted 6 hours before the test. The dog was re-anæsthetized with pentobarbitone and the abdomen re-opened. Diodone was injected down the cannula, and a control film taken. The liver was then tilted from side to side by a hand inserted below the diaphragm. After a suitable interval a further injection of diodone was made into the hepatic vein.

RESULTS

Controls.—Injection of radio-opaque material down the hepatic vein cannula resulted in quick passage of the opaque medium into the inferior vena cava, outlining a length of hepatic vein of about 2·5–5 cm.

Effect of Histamine.—Histamine was given to one dog in doubling doses of 5, 10, 20, 40 μg. histamine acid phosphate. At 40 μg. histamine the hepatic vein in this animal showed slight constriction and at 80 μg. there was a definite constriction (*Figs.* 254, 255). This constriction could not be relieved by instillation of 25 μg. adrenaline down the hepatic vein cannula, but was relieved by infusion of 50 μg. adrenaline. Histamine (80 μg.) caused the constriction of the hepatic vein to reappear. In the three other animals 40 μg.–8 mg. of histamine was administered, causing the hepatic vein to constrict as before. The problem remained as to how much of the hepatic vein was constricted, i.e., did constriction occur distal to the cannula in the non-visualized hepatic veins? In 2 of the dogs injection of diodone was accompanied by narrowing of the hepatic vein and reflux alongside the cannula into tributary veins. The veins so visualized appeared unconstricted (*Fig.* 256). Injection after death revealed wide open hepatic veins and reflux throughout the liver (*Fig.* 257), in contrast to the vasoconstriction which was present immediately before death.

In one of the animals which received 120 μg. histamine (8 μg./kg.) marked narrowing of the

hepatic vein occurred and the lobes drained by the left hepatic vein became opaque to X rays. No other lobes of the liver were thus affected.

Effect of Trauma.—Tilting of the liver from side to side resulted in the liver becoming dark and congested, and the margin of the organ lost its sharpness. The second injection of diodone revealed an obvious constriction of the hepatic vein identical with that seen following histamine administration. The animal died five minutes later and a film at this time showed the hepatic vein to be wide open.

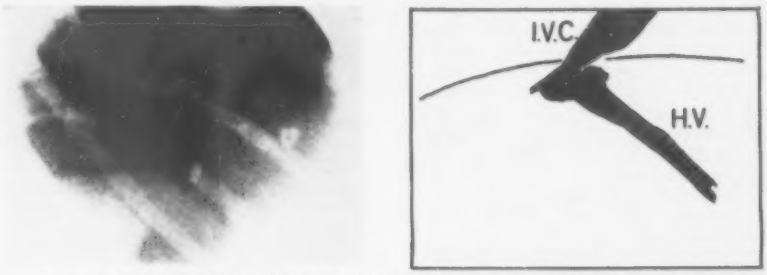

FIG. 254.—Left hepatic vein in a dog outlined by diodone. Control film before histamine injection.

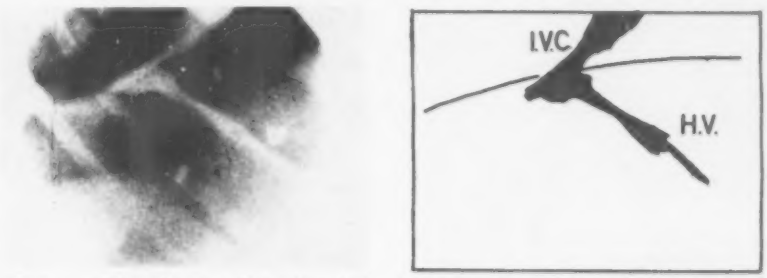

FIG. 255.—Effect of injection of 80 μg. histamine into left hepatic vein followed by 50 per cent diodone.

FIG. 256.—Demonstrates reflex of diodone in the peripheral hepatic veins following constriction of main vein by histamine.

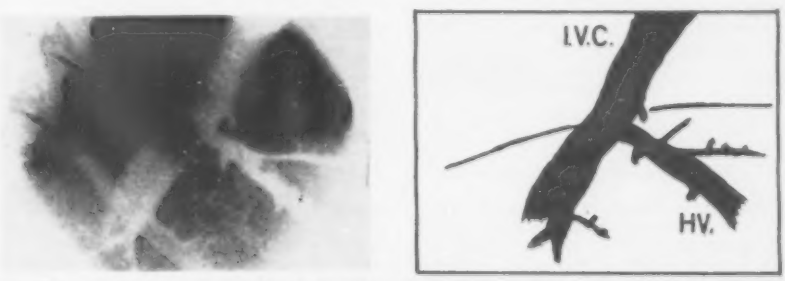

FIG. 257.—Injection of left hepatic vein after death. The vein is widely distended and reflux is seen into the tributaries. Inferior vena cava fills readily.

DISCUSSION

The experiments described above were designed to elucidate whether there exists in the dog a sphincter-like action in the large hepatic veins close to the inferior vena cava.

The radiological appearances of the venous architecture of the liver lobe seen in *Fig.* 253 was obviously due to a hold-up in flow of blood from this lobe and retrograde passage of the radio-opaque material. This back-flow could be due either to complete obstruction of the lumen of the vein by blood-clot, or to a localized constriction of the wall of the vein—a sphincter-like action. The second possibility was suggested by the gradual widening of the hepatic vein in subsequent serial films.

Attempts have been made previously to study the hepatic venous architecture radiologically, by use of a Cournand catheter inserted from the inferior vena cava (Friedman, Frank, and Fine, 1952), or by following the course of the portal injection of the radio-opaque dye (Franklin and Janker, 1937). The former may be criticized because of the length of the catheter in the hepatic venous system which can itself produce spasm of the vein (Bradley, Ingelfinger, Bradley, and Curry, 1945). Friedman and others (1952) also found that withdrawal of such a catheter to the confluence of the lobar veins resulted in poor filling of the hepatic tributaries. Portal injection of dye also results in poor filling of the hepatic veins which are further obscured by the overlying portal veins. The technique used in the present work required only a short length of catheter in a large vein, and injection in the normal direction of the flow. Unfortunately, however, the method can only outline the main left hepatic vein.

The constriction of the hepatic vein observed by us was present in the terminal 1·5–2 cm. of the vein near its junction with the inferior vena cava. This, of course, is the area receiving the maximum dose of histamine and it could be argued that the maximum constriction would necessarily take place there. However, doses of up to 8 mg. histamine were given in a 15-kg. dog without evidence of generalized hepatic vein constriction. In this animal 15 mg. histamine had been injected over the course of 90 min. and the picture was one of constriction of the distal portion of the hepatic vein with a wide portion of vein proximal to this. There was no evidence of widespread hepatic venous constriction.

These results conform with the pressure records made by Bauer and others (1932), who believed that there was a local sphincter action at the entrance of the hepatic vein into the inferior vena cava. They are at variance, however, with the studies of Thomas and Essex (1949), who made casts of the hepatic venous tree following the injection of histamine generally at smaller doses than in the present study. They concluded that their casts showed diffuse spasm of the entire hepatic venous system.

Although histamine is well known to cause constriction of some portion of the hepatic venous system (Andrews, Hecker, Maegraith, and Ritchie, 1953), Friedman and others (1951) were unable to demonstrate this in studies involving retrograde catheterization of the hepatic vein with a cardiac catheter. In their experiments the catheter traversed the area in which we noted constriction to occur.

In the present work a sphincter-like action of the terminal portion of the left hepatic vein was also demonstrated in one dog subjected to hepatic trauma. The effects of trauma and histamine injection appear to be identical, though we are reluctant to draw definite conclusions from one experiment.

SUMMARY

1. Histamine acid phosphate injected into the left hepatic vein of the dog produced constriction of the terminal 1·5–2 cm. of the vein at its entry into the inferior vena cava.

2. Trauma to the liver in one dog produced a similar effect.

3. Radiographic studies suggest that the terminal portion of left hepatic vein has a segmental sphincter action.

We wish to thank Professor D. M. Douglas, of the Department of Surgery, for his advice and help in this work.

REFERENCES

Andrews, W. H. H., Hecker, R., Maegraith, B. G., and Ritchie, H. D. (1953), *J. Physiol.*, **122**, 53.

Bauer, W., Dale, H. H., Poulsson, L. T., and Richards, D. W. (1932), *Ibid.*, **74**, 343.

Bradley, S. E., Ingelfinger, F. J., Bradley, G. P., and Curry, J. J. (1945), *J. clin. Invest.*, **24**, 890.

Deysach, L. J. (1941), *Amer. J. Physiol.*, **132**, 713.

Franklin, K. J., and Janker, R. (1937), *J. Physiol.*, **89**, 160.

Friedman, E. W., Frank, H. A., and Fine, J. (1951), *Ann. Surg.*, **134**, 70.

Gibson, J. B. (1958), M.D. Thesis, Edinburgh.

— — (1959), *J. Anat., Lond.*, **93**, 368.

Knisely, M. H., Harding, F., and Debacker, H. (1957), *Science*, **125**, 1023.

Maegraith, B. G., Andrews, W. H. H., and Wenyon, C. E. M. (1949), *Lancet*, **2**, 56.

Shoemaker, W. C., Walker, W. F., Van Itallie, T. B., and Moore, F. D. (1959), *Amer. J. Physiol.*, **196**, 311.

Thomas, W. D., and Essex, W. E. (1949), *Ibid.*, **158**, 303.

SHORT NOTES OF RARE OR OBSCURE CASES

OBSTRUCTION OF POPLITEAL ARTERY BY SYNOVIAL CYST

REPORT OF A CASE

By DOUGLAS ROBB

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CASE REPORT

Mr. R. A. W., aged 39 years, felt sudden pain in the right ankle while walking uphill in January, 1959. During the next month the pain recurred, sometimes in the ankle, sometimes in the calf, always on effort, and within two months he was reduced by pain to cease walking after 200 yards on the flat. Medical treatment gave no results, and surgery was sought in September, 1959.

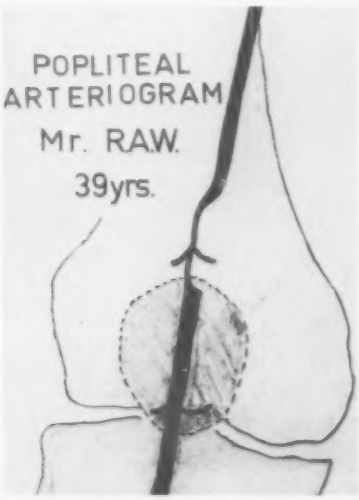

FIG. 258.—Tracing of arteriogram showing narrowed segment in popliteal lumen.

He had worked as a plasterer from 1945 to 1956, and again during the second half of 1958 for six months. In the latter period, almost up to the onset of the pain, he had worked on floors, kneeling on his right knee. The same applied to about 25 per cent of his time in the 11-year period. Since the pain he had driven a small truck around a timber yard.

He was a robust man; he had smoked an average of 4 oz. of cigarette tobacco per week for the previous eighteen years. There were no cardiac symptoms and the E.C.G. was normal. All pulses were palpable, including right popliteal, posterior and anterior tibials, the latter three being markedly diminished. There were no trophic changes in the toes and feet, but after exercise the right toes and foot would become numb and white.

AT OPERATION.—The right femoral arteriogram showed normal vessels with smooth walls, except for the upper popliteal where the lumen was reduced to a thread over 1½ in. by something encroaching from one side. The usual branches of the popliteal were outlined. At exploration a fusiform swelling of the popliteal artery was found as expected, though it lay lower in the fossa than had been suggested by the radiograph. The entering artery was slightly smaller than it might have been and a little thickened in the wall. The swelling was translucent and later this part yielded thick, clear jelly on puncture. It was as if an almond-shaped ganglion had been intimately bound alongside the artery, compressing it. Dense adhesion to adjacent veins was encountered, particularly at the lower end, but no connexion was made out with the knee-joint. The vessels arising from it were divided in turn until at the lower end a normal vessel was seen disappearing between the gastrocnemius heads. It

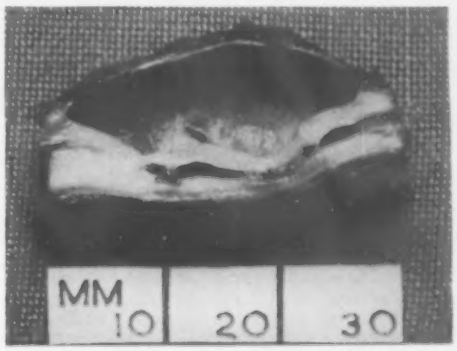

FIG. 259.—Photograph of resected specimen showing empty cyst above and below the artery compressed and partially occluded by thrombus.

FIG. 260.—Microphotograph of wall of artery and cyst—Verhoff elastic Van Gieson stain. *Top left corner*: Flattened endothelium lining cyst over compressed external elastic fibres of the wall of the artery. *Lower right corner*: Internal elastic lamina with attached organized thrombus. (× 135.)

seemed impossible to restore the vessel by evacuating or removing the cyst and this was confirmed by the pathologist, who displayed in addition intramural thrombosis, presumably from pressure. The affected segment, about 2 in. long, was resected, the vessel being cut back a little at each end to remove some thickened intima. A graft from the internal saphenous vein was inserted by end-to-end suture and a good pulse was felt in the graft and in the lower segment on release of the clamps. The vein segment was taken from just above the knee-joint level, but it would have been better to have taken it higher in the thigh, where the bore would have been wider.

Post-operative progress was satisfactory as regards healing and warmth of foot and toes, palpable pulsations at ankle level being disappointing.

PATHOLOGY.—Dr. S. Williams reports as follows:—

Segment of Popliteal Artery.—A segment of artery 3 cm. in length after fixation. It has an external diameter of 0·7 cm. at either end and when cut shows a narrow lumen with an average diameter of 3 mm. Lying along one side of the segment there is a fusiform thin-walled cyst compressing the arterial wall and apparently arising from the adventitia. It measures 1 cm. in diameter at its widest point. It is filled with clear, sticky, mucoid fluid.

Histology.—Sections show the cyst to lie in the adventitia outside the arterial wall proper. It is composed of a thin layer of dense fibrous tissue lined by a single layer of flattened endothelial cells and thus has the structure of a simple synovial cyst. There is no evidence of neoplasia or inflammation. The underlying artery is compressed and shows an old organized and partly recanalized thrombus occluding half the lumen. Otherwise the vascular structure is normal. The origin of this unusual pathological condition is not clear. The most likely explanation is cyst formation from a piece of synovial membrane either from a tendon-sheath or the knee-joint, which has become attached to the artery as a result of trauma or developmental aberration. This has in turn led to partial thrombosis and substantial occlusion of the artery. The specimen has been photographed in longitudinal section.

The illustrations show a tracing of the arteriogram (*Fig.* 258), a photograph of the specimen cut longitudinally (*Fig.* 259), and a photomicrograph of the vessel wall (*Fig.* 260).

DISCUSSION

The nature of this lesion seems clear enough, though the intimacy of the relation of the cyst to the wall of the artery is difficult to explain. Such cysts of course are common in this region, but are often connected with the joint or lie free. Mr. Wade, of the Library, Royal Society of Medicine, who kindly searched the literature for me, was unable to refer to a comparable situation. Burleson, Bickel, and Dahlin (1956), in discussing popliteal cyst, made no mention of affection of the artery. Fienberg and Baehr (1941) described a hæmangio-endothelioma of the tibia which metastasized to the popliteal artery, and Neighbours and Green (1952) an occlusion of the popliteal artery from organized venous clot.

REFERENCES

BURLESON, R. J., BICKEL, W. H., and DAHLIN, D. C. (1956), *J. Bone Jt Surg.*, **38A**, 1265.

FIENBERG, R., and BAEHR, F. H. (1941), *Arch. Path.* (*Lab. Med.*), **31**, 811.

NEIGHBOURS, J. B., and GREEN, J. A. (1952), *J. Med. Ass. Georgia*, **41**, 97.

MULTIPLE INTUSSUSCEPTION AND INTESTINAL PERFORATION DUE TO A BEZOAR

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THE occurrence of trichobezoars is unusual but well known. The symptoms they produce tend to be vague, consisting mainly of epigastric discomfort, anorexia, and vomiting. Their presence may be revealed by the palpation of an epigastric mass, by barium-meal examination, or at laparotomy. The occurrence of a case of a previously unsuspected bezoar causing multiple intussusceptions and intestinal perforation seems sufficiently interesting to report.

CASE REPORT

Marilyn was a girl 10 years of age when she was admitted on April 18, 1959, to hospital near her home. She was complaining of a five-day history of generalized mild abdominal pain which had increased over the past few hours and had in this time been accompanied by vomiting. She had not had a bowel action for three days prior to her admission. At examination she was noted to be pale and miserable; she had a dirty tongue with considerable fœtor but there was no pyrexia. There was generalized mild tenderness on abdominal palpation but no other abnormality. The following day her general condition remained the same but her abdomen became a little distended and she developed a pyrexia of 100° F.

On April 20 the abdominal distension and tenderness had increased, the pyrexia continued, and the pulse-rate became more rapid. As the clinical signs suggested acute appendicitis laparotomy was performed through a gridiron approach. Six intussusceptions of the small intestine were found, all of which reduced readily. There was a great number of enlarged mesenteric glands and it was noted that the mesenteric border of the small intestine felt thickened. The appendix appeared engorged, and was removed. Post-operatively her progress was slow and on April 24 she was reported to have vomited a "large piece of fæces". As at this time her abdomen was still distended a diagnosis of post-operative intestinal obstruction was made and nasogastric suction and intravenous therapy were commenced. A white blood-cell count showed 6000 cells per c.mm. with a normal differential reading, and a straight radiograph of the abdomen showed fluid levels in distended small intestine.

On the evening of April 24 she was transferred to the Hospital for Sick Children, Great Ormond Street. On admission there she was seen to be a quiet, obviously ill child. Her tongue was thickly coated and she had foul

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fœtor. Her abdomen was mildly distended and tender and a full bladder was easily palpable. Rectal examination was normal.

It was decided to observe her response to conservative therapy overnight. By the following morning, as the abdominal distension and tenderness had increased and her general condition deteriorated, laparotomy again became necessary. This was performed through a right paramedian incision, and the peritoneal cavity was seen

FIG. 261.—The bezoar, with its extension into the duodenum, and part of the strand of wool which extended the length of the small intestine.

to contain a moderate amount of straw-coloured free fluid; several distended loops of small intestine presented in the wound. These loops, which were of terminal ileum, were withdrawn and four intussusceptions were seen, one being in a retrograde direction, and the longest involving about 12 cm. of bowel. All of these were reduced without difficulty and the bowel wall did not appear unduly œdematous. The large number of mesenteric glands previously noted was seen and also the thickened mesenteric border of the gut. The ileum was then inspected for evidence of multiple tumours to try to account for the intussusceptions, and through the ileal wall dark shadows were seen which were mobile for about 1 cm. in the long axis of the bowel. The ileum was therefore opened along its antimesenteric border; the 'tumours' were seen to be small masses of matted wool attached to a longitudinal strand of wool which on further examination proved to extend the whole length of the duodenum, jejunum, and ileum from a bezoar in the stomach. This wool, which had obviously been 'woven' into a strand by peristaltic action, had had the effect of greatly foreshortening the mesenteric border of the gut, thus producing multiple intussusceptions commencing initially in the antimesenteric border. The thickening was seen to be œdema around a linear ulcer of the mucosa extending the whole length of the mesenteric border of the small intestine secondary to pressure caused by this tight band. Inspection of the duodenojejunal flexure showed that at this fixed point the ulcer had recently perforated.

Following extension of the skin incision five enterotomies were made to remove, in lengths, this taut strand from the small bowel. Gastrotomy was then performed and the bezoar removed together with its extension into the duodenum (*Fig.* 261). A nasogastric tube was then passed through the mouth into the stomach and thence threaded through the duodenum to the jejunum beyond the perforation. The duodenojejunal flexure was mobilized as much as possible and the perforation in the very œdematous bowel wall closed with a layer of continuous catgut using a Connell stitch. This was reinforced by oversewing omentum and a corrugated rubber drain placed down to this level through a stab incision. After closure of the gastrotomy the skin incision was closed in layers. A second nasogastric tube was inserted into the stomach itself.

The child's condition at the end of the operation was very poor. During the operation 540 ml. of blood and 400 ml. of plasma were given, together with 100 mg. intravenous hydrocortisone immediately afterwards. The blood-pressure following this slowly improved. Intramuscular hydrocortisone was administered in diminishing doses over the next three days and parenteral tetracycline therapy continued for six days. Continuous suction was applied to the nasogastric tube and also to the jejunal tube until it was vomited on the second day. All the fluid aspirated was measured and samples sent for analysis. On the results of these investigations and of serum electrolyte readings, fluid and electrolyte replacement was planned.

On the fifth day bowel-sounds became audible, and on the sixth day the nasogastric tube was removed. Following this she made an uninterrupted recovery and was discharged home well on the fifteenth post-operative day.

DISCUSSION

Quite apart from the interesting diagnostic problem posed by this child, no other similar case has been found in a search of the literature. Multiple intussusceptions are uncommon and may be caused by multiple intestinal tumours, gastro-enteritis, purging, or abdominal trauma. Small ones have been seen occasionally as an incidental finding at laparotomy in childhood, and they have also been found at post-mortem examination. Their occurrence with no apparent cause has been ascribed to some neuromuscular incoordination. This child's intussusceptions were undoubtedly caused by the mechanical foreshortening of the mesenteric border of her gut combined with its œdema, allowing the normal antimesenteric border to fold upon itself and thus commence an intussusception.

On reconsidering the child's history after the operation it was recollected that the mother had said that her daughter had been in the habit of plucking her jerseys, but the significance of this had not been realized at the time. As she became well post-operatively Marilyn seemed to be a rather quiet but pleasant little girl of normal intelligence. The cause of all her trouble was explained to her before she was discharged and when she was seen again in Out-patients several weeks later she had apparently overcome her wool-plucking habit and appeared to be very well.

I would like to thank Mr. G. H. Macnab not only for enabling me to look after this child, but also for his advice in the preparation of this paper.

SQUAMOUS CARCINOMA ARISING IN AN ANAL FISTULA

By J. C. MILLIKEN, Belfast

It has long been realized that malignant degeneration may occur as a sequel to many long-standing irritative lesions, and instances of the development of cancer in anal fistulæ have been recorded by Yeomans (1929), Rosser (1931), Ducassi and Smith (1945), McIntyre (1952), and others. However, the condition is uncommon and recent references to it in British literature (Doran, 1948; Dukes and Galvin, 1956) are few. In most of the recorded cases the growth has taken the form of an adenocarcinoma or colloid carcinoma, and instances of the occurrence of a squamous-celled tumour are extremely rare.

CASE REPORT

Mr. B. M., aged 61 years, was admitted to hospital on Sept. 2, 1958, complaining of painful swelling of his left ischiorectal region of two days' duration. Twenty years previously he had had an operation for a left ischiorectal abscess, and since then he had had a persistent discharge from several chronic sinuses in that area. Two months before admission he noticed a warty growth at the site of the discharge, and this had continued to enlarge steadily.

On examination there was a red indurated swelling of the left ischiorectal region, on the surface of which were multiple sinus openings. Immediately adjacent to the most lateral of these was a papillomatous growth about 1½ in. in diameter. Rectal examination revealed moderate tenderness on firm pressure to the left, but otherwise was negative. One left inguinal gland was palpable.

There was no family history of tuberculosis, and radiographs of chest and spine were normal. The white-cell count was 6150, and on culture of the discharge *Staph. aureus* and *B. proteus* were isolated. Subsequent histological examination of the sinus showed no evidence of tuberculosis.

The growth and the contents of the ischiorectal fossa, which consisted of indurated tissue containing sinuses and small abscesses, were excised. No internal opening into the rectum was demonstrated. The resulting cavity was allowed to heal by granulation. Healing was uneventful, and three months later the inguinal gland was no longer palpable. At review in October, 1959, there had been no recurrence of the discharge or of the growth, nor was there any evidence of a tumour in the rectum.

Pathological examination of the excised tissue showed a papillary-like growth in relation to a sinus tract showing chronic non-specific inflammation with marked hyperplasia of epithelial elements. The histological appearances of the tumour were those of a squamous carcinoma, and the pathologist concluded that it had supervened on the long-standing inflammatory changes.

DISCUSSION

Three possibilities must be considered in determining the mode of origin of cancer in an anal fistula.

It has been shown that cancer cells shed from a growth at a higher level in the bowel may become implanted as secondary deposits in a pre-existing fistula (Guiss, 1954). Such deposits will retain the adenocarcinomatous nature of their primary site.

Dukes and Galvin (1956), in a review of 10 cases, in 8 of which colloid carcinomata developed in fistulous tracts, postulated that these were, in fact, cases of carcinoma occurring in congenital malformations of the lower end of the alimentary canal, as detailed study of the fistulæ had shown some of them to be lined with normal mucus-secreting epithelium, with a well-defined muscularis mucosal layer. Again, this explanation can be applied reasonably to cases with columnar-celled growths only.

Rosser (1934) suggested that the following criteria should be fulfilled in order to accept that malignant degeneration had occurred in an anal fistula:—

1. The history of the fistula should be sufficiently long to exclude the possibility of the carcinoma having been the original lesion.
2. No tumour should be present in the rectum except as a definite secondary extension.
3. The internal orifice of the fistula should be in an anal crypt or ulcer, but not in malignant tissue.

The case described here satisfies these requirements. As with Doran's (1948) patient, no internal orifice could be demonstrated, but the malignant change was confined to the external opening of the fistula, and even after more than a year no primary rectal growth was found. There appears to be no doubt that the neoplastic degeneration occurred in response to the chronic irritation of the fistulous discharge.

SUMMARY

A case is presented of carcinoma developing in an anal fistula of twenty years' duration. Usually such growths are adenocarcinomatous, and may possibly occur by implantation of cells from a primary growth at a higher level in the bowel. In this case the tumour was squamous celled, and followed long-standing inflammation.

I wish to thank Mr. H. W. Gallagher, Consultant Surgeon, Banbridge Hospital, for his help and permission to publish this case report, and Dr. J. E. Morison, of the Central Pathology Laboratory, Belfast, for the pathological report and for encouragement. I am also indebted to Professor H. W. Rodgers for his advice.

REFERENCES

Doran, F. S. A. (1948), *Brit. J. Surg.*, **36**, 100.
Ducassi, E. R., and Smith, N. D. (1945), *Proc. Mayo Clin.*, **20**, 57.
Dukes, C. E., and Galvin, C. (1956), *Ann. R. Coll. Surg. Engl.*, **18**, 246.
Guiss, R. (1954), *Surgery*, **36**, 136.
McIntyre, J. M. (1952), *Amer. J. Surg.*, **84**, 610.
Rosser, C. (1931), *Ibid.*, **11**, 328.
— — (1934), *Trans. Amer. proct. Soc.*, **35**, 65.
Yeomans, F. C. (1929), *Proctology*, 495. New York: Appleton.

PRIMARY VOLVULUS OF THE SMALL INTESTINE IN AN ADULT

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SPONTANEOUS small-intestinal volvulus is common in some vegetarian populations in Africa (Kerr and Kirkaldy-Willis, 1946) and in India (Banerji, 1951) in districts where a bulky farinaceous diet is taken. This precipitating factor is absent in European countries and the condition of spontaneous (primary) volvulus of this reach of the intestine is stated to be extremely rare (Aird, 1957). Only 3 of 37 cases of small-bowel volvulus at the Mayo Clinic were primary (McKechnie and Priestley, 1936). It is felt, therefore, that the following case report may be of interest.

CASE REPORT

A. H., a 28-year-old married woman, was at the cinema when she experienced a lower abdominal cramp which increased in severity and necessitated her leaving and walking one mile to her home. The cramp subsided for an hour or two but then returned with great severity and her doctor arranged for her examination at the Casualty Department of Glasgow Royal Infirmary. Her history revealed that she had missed one menstrual period and was fourteen days overdue. She had not suffered from nausea and had not vomited. A non-tender cystic swelling about the size of an orange was palpable in her pelvis but did not appear to be intimately attached to her uterus. There was no muscle guarding or distension. Her blood-pressure was 120/80 and her pulse-rate 80 beats per minute. It was decided to admit her to a gynæcological ward for observation as a suspected ruptured ectopic pregnancy or a twisted ovarian cyst. In the course of the following twelve hours her abdominal pain decreased and finally disappeared. A repeated physical examination failed to identify any trace of the cystic swelling. At the end of this interval her spasms of pain returned with increasing severity and the cystic mass became palpable again, this time being greater in size, tender, and accompanied by lower abdominal muscle guarding. Her blood-pressure and pulse-rate remained unchanged. It was now considered that she was a case of ruptured ectopic gestation, but that the bleeding was into the broad ligament and limited. At this stage she vomited for the first time.

OPERATION FINDINGS.—Laparotomy was performed through a lower midline incision and immediately the peritoneum was incised a loop of gangrenous small bowel prolapsed and a quantity of blood-stained exudate escaped. The lesion was found to be a volvulus of the small intestine, 12 in. (30 cm.) from the ileocæcal junction. The loop measured 14 in. (35 cm.) and the twist was two and a half turns in an anticlockwise direction. There was neither distension of the afferent limb nor collapse of the efferent. The volvulus was reduced by rotating the loop in a clockwise direction and the root of the mesentery was found to be of normal length, about 6 in. (15 cm.). There was no obvious redundancy of small bowel or evidence of adhesion, diverticulum, or tumour. The gangrenous loop was resected, the small intestine reconstituted by axial anastomosis, and the abdomen closed without drainage. The excised loop was opened and examined and, other than the changes associated with strangulation, no abnormality was apparent.

POST-OPERATIVE TREATMENT.—The patient received one bottle of compatible blood and four bottles of intravenous 5 per cent dextrose in distilled water. Aureomycin was administered in 250-mg. doses in each of the four bottles. She made an uninterrupted recovery and was discharged on the fourteenth post-operative day. At her subsequent attendances as an out-patient she was fit and well.

DISCUSSION

Volvulus of the intestine is generally considered to be caused by some initiating irritation acting upon a part of which the mesentery is sufficiently elongated to allow the extremities of a loop to approximate to one another. Neither of these features was evident, and interrogation of the patient failed to reveal any dietary indiscretion or the use of any aperient which might have initiated the lesion.

It is of note that this case simulated a gynæcological emergency, and in a similar case Hamman (1951) pointed out that the presence of signs of blood-loss (due to blood-stained transudate) and the finding of a cystic tumour (the volvulus) on vaginal examination would suggest an ectopic gestation or hæmorrhage into an ovarian cyst in the absence of obstructive signs. The suspicion raised by these, as would be expected from the proximal occlusion, would indicate a straight abdominal X-ray plate, which would establish the diagnosis. Such an investigation was not considered in this case.

The remission of the symptoms and signs which occurred was almost certainly due to the spontaneous reduction of an intermittent partial volvulus. This phenomenon has been previously reported by Salisbury Woods (1945), where, to quote his apt description, "the affected loop was swinging like a ship at anchor, partially folding over and then swinging free again until it 'stayed put'".

SUMMARY

A case of the rare primary volvulus of the small intestine is reported and attention is drawn to its simulation of a gynæcological emergency.

I wish to acknowledge my indebtedness to Mr. William Patrick, Glasgow Royal Infirmary, in whose care this patient was, for permission to publish the report.

REFERENCES

AIRD, I. (1957), *Companion in Surgical Studies*, 2nd ed., 822. Edinburgh: Livingstone.

BANERJI, B. N. (1951), *Indian J. Surg.*, **13**, 311.

HAMMAN, J. (1951), *S. Afr. med. J.*, **25**, 241.

KERR, W. G., and KIRKALDY-WILLIS, W. H. (1946), *Brit. med. J.*, **1**, 799.

MCKECHNIE, R. E., and PRIESTLEY, J. T. (1936), *Amer. J. Surg.*, **34**, 286.

WOODS, R. S. (1945), *Brit. med. J.*, **2**, 496.

REVIEWS AND NOTICES OF BOOKS

The Royal College of Surgeons of England: A History. By ZACHARY COPE, Kt., Fellow of the College. 8¾ × 6¾ in. Pp. 360, with 78 illustrations. 1960. London: Anthony Blond, Ltd. 63s.

ON the title-page of this eagerly anticipated volume, the author modestly presents himself as Zachary Cope, Kt., Fellow of the College. Known to generations of London students as a genial surgeon and teacher, as the skilled biographer of William Cheselden and Florence Nightingale, the historian of his (St. Mary's) Hospital, and the troubadour of abdominal emergencies, Sir Zachary has undertaken with rare courage and devotion a highly difficult task in presenting *A History* of the College. (His use of the indefinite article is further evidence of his innate modesty.)

It has been a truly formidable task. John Flint South, of St. Thomas's, had spent 20 years gathering the materials for his unfinished *Craft of Surgery* (1886). He, too, had found it difficult to "reconcile the need for historical accuracy with the natural desire to write a readable story". But captious criticism has no right of place here as we read a story every page of which bears evidence that for the writer this has been a labour of love.

The present reviewer has read the *History* twice through, from cover to cover. On first reading he was, at times, guilty of impatience over this detail or that; but, having left it aside for more pressing duties, he came back, as it were, with an 'informed eye', and now regards it as an intensely dramatic presentation of four centuries of history in three acts. The Fellows and Members of the College to-day, rightly and proudly, trace their descent unbroken from Tudor days when, as depicted in Holbein's great canvas, Thomas Vicary and his Company knelt to receive their first Charter of Incorporation from Henry VIII (1540). Against this backcloth we follow the play of the guildsmen who with others of their kind were narrow-minded believers in the 'closed shop', and whose public spirit, in the words of a later commentator, "was sometimes more than questionable". History has been ungenerous to these humble followers of Vicary. By comparison with the physicians of their day they were men of a lower social order, yet there were amongst them men of equal stature to that of an Ambroise Paré or a Georges Mareschal across the Channel. Cheselden, we recall, was the first to be chosen by Mareschal as an *associé étranger* of his *Académie* of 1731, and to Cheselden's urge for the teaching of anatomy the London surgeons owed the liberation of the new Company from the control of the City authorities. Yet with their newly-won freedom they still held to the apprentice system, the Master with his two Wardens (forerunners of the President and his two Vice-Presidents of the present day) and their Court of Assistants. The new system carried too many loopholes for official sinning, and it needed a Gunning's philippic to call them to order, as he did in 1790. With a new Charter granted by George III in 1800, the Royal College of Surgeons of London was to fall into line with the sister Colleges of Edinburgh (1778) and Dublin (1784).

The second act covers the longest spell of years, opening and closing to the 'alarums and excursions without' of two European wars. Pitt may have had little money with which to buy gunpowder, but his Government, in handing over John Hunter's biological collection to the care of the new College, made a handsome contribution towards the building of a Museum fit to house it. (Later in the *History* the present Curator, Miss Jessie Dobson, gives a succinct and admirably clear account of the development of the Museum and its century-long stimulus to research and teaching since its early development under the care of Clift and Owens. In her *Life of William Clift* (1954) she has immortalized for future generations the selfless devotion of the man whose six unpaid years of care after Hunter's death had preserved this priceless collection for posterity.)

There follow then the troublesome years of 'reform' within the College administration, years made noisy with the claims of the dissatisfied Members and the crude journalism of the egregious Wakley. Many of the complaints against the Council were well founded. By the middle of the century the birth of the B.M.A., the establishment of the Medical Register under the care of the General Medical Council, and the later admission of women to Membership of the College signalized the defeat of the 'die-hards' of the old régime in the slow evolution of the Conjoint Board, with a new Examination Hall on the Embankment as the visible sign of timely advancement in the surgeon's education. The curtain falls on the celebrations for the centenary of the Charter of 1800. In the latter half of the century had come a succession of gifts in anæsthesia, in Lister's adoption of Pasteur's work, Roentgen's X rays, and the Curies' radium, with Ehrlich's magic bullet of chemotherapy, all of which, in Moynihan's lapidary phrase, "had made surgery safe for the patient".

History, Carlyle has told us, is but the shadow cast by a few great men. As our curtain rises on Act III there steps on to the stage a fortunate succession of men who, as Presidents, have raised the College to its present position of eminence in our surgical education. Many of us have known them personally and, in reading Sir Zachary's account of the past half-century, will agree that he has kept the best wine to the last.

Moynihan had breathed a new spirit into the Council: his clarion call to "make the patient safe for surgery" rang around the English-speaking world, and the launching of the *British Journal* gave British surgeons a window in which to set their new gospel of physiological fitness; with his travelling club's journeys at home and abroad he broke fresh ground for exploration. The surgeon was no longer the unlettered man his forebears had been, and Moynihan's personality drew to the College many friends to support the new outlook. After him came Webb-Johnson, with his experience and foresight in the hour of gravest need to seize opportunity and find the necessary funds to rebuild and expand the burnt-out palladium of British surgery. His years of leadership—in which he was ably backed by friends both within and without the College—saw it rise, Phœnix-like, from its ashes till to-day, as yet incomplete, it stands pre-eminent as a post-graduate centre of teaching and research. To Lincoln's Inn Fields the younger surgeons of the Commonwealth turn to-day as a Mecca, there to find Hunter's Museum renovated and enriched and a Library such as Gunning had never dreamed of. The final curtain has not fallen yet, and the newer players crowd the stage to bring its drama to greater heights as yet unseen—perhaps unforeseeable.

Sir Zachary has left his College deeply in his debt. In such a story-telling we are blind to the occasional *longueur*; but with one small sin of omission we must charge him. Heraldry, like surgery, is both Art and Science. We would gladly have given the tale of the Storms in the Teacups of Chapter 9—a vicarious intromission in such a history—for an account interpreting for us the Coat of Arms emblazoned on the publisher's cover. For is not heraldry the romantic footnote to the story of arms and the men who bore them?

Clinical Neuro-Surgery. Volume 6. Proceedings of the American Congress of Neurological Surgeons, San Francisco. 1958. 9 × 5¾ in. Pp. 289 + xx, with 120 illustrations. 1959. Baltimore: The Williams & Wilkins Co. (London: Baillière, Tindall & Cox.) 64s.

THIS is the sixth of these volumes, which, being alike in size and shape, make a handsome collection as they accumulate. As they have appeared the previous ones have been reviewed in this JOURNAL, the first in May, 1956, the last in May of last year. The present volume consists of the edited proceedings of the eighth annual meeting of the Congress of Neurological Surgeons which was held in San Francisco towards the end of 1958. It is dedicated to A. Earl Walker, about whom there is a bibliographical sketch by R. G. Fisher, followed by a list of his 204 contributions to his subject. Earl Walker himself contributes three chapters on, respectively, the dawn of neurosurgery, the surgery of the brain-stem, and post-traumatic epilepsy. Altogether there are fifteen separate chapters, mostly on intracranial problems, but there is one on syringomyelia and one on the biochemical aspects of space flight by A. W. Hetherington, who concludes a scholarly summary of our present knowledge of the problems concerned with space travel by casting some doubt on its feasibility because of the unknown effects of cosmotic radiations, of weightlessness, and on the mind, but in the last connexion he quotes Air Force psychiatrists who believe that space flight should impose no intolerable stress on carefully selected and trained crews.

Like the preceding volumes in the series, this is a valuable record of some of the problems of modern neurosurgery and the efforts which are being made for their solution.

Klinik und Praxis der Urologie. By WERNER STAEHLER. Volume II. 10½ × 7½ in. Pp. 863 + xxii, with 641 illustrations. 1959. Stuttgart: Georg Thieme. D.M.240.

THE appearance of this comprehensive treatise underlines the fact that Urology is a full medical specialty, and finally inters the idea that it is but applied plumbery.

Contrary to fashion, its authorship is undivided, in itself an outstanding feat. The systematic presentation of the material, the many apposite case-histories and high-quality illustrations, the excellent operative section, and the widely representative bibliography all deserve high praise. Having sketched the skeleton of the work, an attempt is made to sample the meat.

Practical variation from what is usage in this country is inevitable but unimportant. Certain differences in emphasis, however, merit some consideration:—

Partial nephrectomy, freely used in the treatment of renal tuberculosis, is applied with caution, not to say reluctance, in renal lithiasis, probably because this condition is viewed more as a metabolic than a local problem.

There is no reservation in recommending enlargement of the contracted tuberculous bladder with an isolated loop of gut; the bogy of tuberculous tissue being inoperable is being laid.

Hypertension due to unilateral renal disease is regarded with Homer Smith's scepticism, not shared by some observers.

Percutaneous renal biopsy is pilloried as a dangerous procedure; it must be utilized with discretion, but it has facilitated the diagnosis of, and certain aetiological studies in, chronic inflammatory and other diseases of the kidney.

Some criticisms may be made: the section dealing with the neurogenic bladder lacks clarity in management. The merit of Ludwig Guttmann's work on this subject lay in unambiguously defining as the aim efficient bladder-automatism; his methods of achieving this are worth drawing on.

The discussion of anuria and oliguria omits mention of the physiological post-operative retention of water (and sodium).

The role of varicocele and hydrocele in male subfertility has been overlooked.

Surgical Philosophy in Mass Casualty Management. By WARNER F. BOWERS, A.B., B.Sc., M.D., M.S., Ph.D., and CARL W. HUGHES, A.B., M.D., Chief and Assistant Chief respectively, Department of Surgery, Tripler U.S. Army Hospital, Honolulu, Hawaii. 8½ × 5½ in. Pp. 204. 1960. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 54s.

THIS is a useful book and appears at an opportune time, for not only does it discuss the effects of modern warfare, but also chemical and bacteriological warfare. The authors have wisely included the lessons learnt from civilian disasters, such as a train wreck, an earthquake, a flood, and explosions by fire. All these things have been all too common during the last year.

Irradiation injury is given its place, and the authors condemn, quite rightly, the publicity that is given to-day to people who persist in speaking outside of their field of knowledge.

This is a book that all doctors should read in this modern age.

Nouvelle Pratique Chirurgicale Illustrée. By JEAN QUÉNU. Part XV. 10½ × 7 in. Pp. 286, fully illustrated. 1960. Paris: G. Doin & Cie. N.F.39·00.

LIKE its predecessors, this fifteenth volume illustrates a random half-dozen operations, each by a few pages of letterpress and a series of detailed drawings.

For this volume the operations selected are:—

1. Removal of an intrathoracic neurofibroma (Georges Thomeret).
2. Operation for anal incontinence using the Gracilis (Claude Dubost).
3. Cysto-jejunostomy "en Y" for pancreatic pseudocyst (Jean Perrotin).
4. Myomectomy for uterine fibroids (Jean Quénu).
5. Colo-cystoplasty for contracted tuberculous bladder (Louis Quénu).
6. Palmar fasciectomy for Dupuytren's contracture (François Dubois).

There is a plethora of full-page drawings (244 in all) illustrating each operation in all its stages, including incidental steps such as the repair of an opening accidentally made in the small bowel. In spite of the clarity and artistic excellence of the illustrations such superfluity of detail becomes a little irritating and is no doubt responsible for the high price of the book. Nevertheless the authors' methods (which are sound standard ones in the main) are well explained by this presentation.

Crash Injuries. By JACOB KULOWSKI, M.D., F.A.C.S., F.I.C.S. 9 × 5¾ in. Pp. 1080 + xlii, with 533 illustrations. 1960. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) £13.

THIS is a large, comprehensive monograph dealing with the integrated medical aspects of motor-car injuries and deaths.

The author has managed to illustrate his work, not only with clinical and pathological pictures showing the effects of trauma, but he has added numerous pictures showing actual crashed cars, and the damage inflicted on them.

Quite a large part of the book is devoted to the treatment of the injured person and the various techniques that are in vogue in the United States of America. The actual death-rate from motor-car accidents is higher in

America than in this country. The autopsy material is enormous and has been used to good advantage in this book.

Safety-belts appear to be little used, but there can be no doubt that such belts do prevent a large number of brain injuries.

The book is well illustrated with numerous photographs, charts and sketches, which do enhance the value of the text.

An excellent reference volume dealing with crash injuries.

Surgical Gastroenterology. By WARNER F. BOWERS, M.D. 9¼ × 6¾ in. Pp. 498 + xii, with 235 illustrations. 1960. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) £7 8s.

TO-DAY the surgical speciality of gastro-enterology has come into its own; even in England a new surgical journal by the apt name of *Gut* has been launched this year.

Dr. Bowers has produced a large and comprehensive volume which deals with the symptoms and treatment of diseases of the gastro-intestinal tract. The surgical techniques are well described and illustrated, and the various pre- and post-operative procedures are given in detail.

A bibliography is included at the end of each chapter.

Care of the Surgical Patient. By J. A. GLASSMAN, Assistant Clinical Professor of Surgery, University of Miami; and RAYMOND W. MCNEALY. 8½ × 6 in. Pp. 320, with 2 illustrations. 1959. Baltimore: The Williams & Wilkins Co. (London: Baillière, Tindall & Cox.) 52s.

THIS is a useful pocket-book for residents and registrars, for it gives in a direct and definite way the various pre- and post-operative treatments which are required in modern surgery to-day.

There are some twelve contributors, each of whom has written upon his own particular subject.

Various charts are given, such as the pyloric balance chart, and tables of normal laboratory values which are so difficult to remember in detail.

A detailed knowledge of this book will be invaluable to any resident surgical officer.

Antibiotics and Sulphonamides in Ophthalmology. By ARNOLD SERSBY and JOSEPH UNGEN. 6¼ × 4 in. Pp. 117 + x. 1960. London: Oxford University Press. 12s. 6d.

THIS is a useful little book, which provides in a small compass a complete and up-to-date guide to present-day practice in the treatment of ocular infections.

The book is divided into three parts: Part I discusses basic problems, infection, and specific diagnosis; Part II deals with the pharmacological and therapeutic aspects of each antibiotic in turn, so far as they are relevant to ophthalmology; Part III describes the specific treatment of ocular infections.

There are many useful tables setting out the dosage for different infections.

An excellent little book which should prove of real value to both students and practitioners.

Fellowship of Surgeons. By LOYAL DAVIS, M.D., F.A.C.S. 9 × 6 in. Pp. 523 + vi. 1960. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 84s.

THIS is a book which is long overdue, but now it has appeared it will prove of great interest to all surgeons on both sides of the Atlantic.

Loyal Davis has written a delightful book, full of interest and history. Revealed for the first time are the thoughts, motives, and actions of famous men of surgery from the inception of the Clinical Congress of Surgeons of North America, including the founding of the College in 1913, up to the present time.

The American College of Surgeons in its inception was intimately associated with the journal known as *Surgery, Gynecology and Obstetrics*, which became its mouthpiece, and has continued ever since to be the official journal of the College.

The author of this excellent volume has included letters from surgeons where they play a part in the history of the College. One of these by Harvey Cushing is quite fascinating.

The American College of Surgeons have had their difficulties in the past in trying to elevate standards of practice and patient care. The differences in outlook between the American College of Surgeons and the International College of Surgeons are well portrayed.

Loyal Davis has performed a great task, no doubt a labour of love, for which we are truly thankful.

Cancer and Allied Diseases of Infancy and Childhood. Edited by IRVING M. ARIEL, M.D., Associate Professor of Clinical Surgery, New York Medical College; and GEORGE T. PACK, M.D., Associate Professor of Clinical Surgery, Cornell University Medical College. 9¼ × 6½ in. Pp. 605 + xiv, fully illustrated. 1960. London: J. & A. Churchill Ltd. 160s.

THIS is a large monograph contributed by some twenty-seven contributing authors, all specialists in their own particular fields of surgery, pediatrics, or pathology.

At first sight it might seem curious that such a book as this is required to be written. However, it must be remembered that cancer is a great killer of infants and children. Cancer is the most frequent cause of death, exclusive of accidents, between the ages of 5 and 25 years. This surely justifies the publication of this book.

The authors cover in a most comprehensive manner all the various tumours which are found in the tissues and organs of infants and children. The chapters on leukæmia and lymphoma are outstanding; so are the chapters on tumours of the central nervous system.

The whole volume is beautifully illustrated by clinical pictures, radiographs, pathological specimens, and photomicrographs.

An up-to-date and extensive bibliography is given at the end of the book.

An excellent reference volume.

First Aid: Diagnosis and Management. By WARREN H. COLE, M.D., F.A.C.S., and CHARLES B. PUESTOW, M.D., F.A.C.S. 8½ × 5½ in. Pp. 420 + xii, with 113 illustrations. 1960. New York: Appleton-Century-Crofts, Inc. $6.25.

THE fifth edition of this popular manual of first aid has been very extensively revised, and many new illustrations have been incorporated in the text.

Warren Cole is to be congratulated on the way he has selected his sixteen contributing authors, for the whole book reads well and there are no disjointed sections.

This is probably the most comprehensive first-aid book yet published, and the practitioner will find useful advice regarding snake-bites, burns, food poisoning, industrial injuries, air-sickness, medical emergencies, and military casualties.

The chapter on respiratory emergencies and respiratory arrest is outstanding.

A very useful book in the present traumatic age.

Thymectomy for Myasthenia Gravis. By HENRY R. VIETS, M.D., and ROBERT S. SCHWAB, M.D. 9 × 5½ in. Pp. 143 + xvi. Illustrated. 1960. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications). 56s.

THIS excellent monograph is a record of experiences at the Massachusetts General Hospital in Boston. The

authors and their associates have performed some 139 thymectomies in the Myasthenia Gravis Clinic which was first established over twenty-five years ago. A careful analysis of these patients has led to the conviction that thymectomy is an important form of treatment in this disease. With proper anæsthesia and careful selection, the operative mortality has been reduced to zero. This is a great achievement.

This book sets out in great detail the operative technique in thymectomy, and describes the favourable types of anæsthesia which should be used in this operation.

Chirurgie de l'Etage Supérieur de l'Abdomen. By HENRI FRUCHAUD. 9½ × 6 in. Pp. 200, with 92 illustrations. 1960. Paris: G. Doin & Cie. N.F.32·00.

THIS monograph describes in detail the anatomical changes in the upper abdomen of man which have evolved as the result of his adoption of the upright stance. These changes are twofold; the roof of the diaphragm has come to lie more deeply than in the quadrupeds and peritoneal bands have formed which plaster the upper abdominal viscera to the posterior abdominal wall.

The authors commend two manœuvres in operations upon the upper abdomen which are based on these changes. The first is to force the diaphragm into a more accessible position by a combination of a high bridge beneath the dorsilumbar spine, to produce a marked lordosis, coupled with a mechanical retractor to pull up the lower costal margins. The second is the adequate preliminary mobilization of the abdominal viscera by freeing their peritoneal attachments.

With the latter no one could find fault; with the former one wonders how bitterly patients might complain of backache and chest pain after having been operated upon in this position.

Year Book of Neurology, Psychiatry and Neurosurgery. Edited by ROLAND P. MACKAY, M.D., Professor of Neurology, University of Illinois, Attending Neurologist, St. Luke's Hospital, Chicago; S. BERNARD WORTIS, M.D., Dean, New York University Schools of Medicine, Professor and Chairman, Department of Psychiatry and Neurology, and Director, Psychiatric and Neurologic Services, New York University Hospital; and OSCAR SUGAR, M.D., Professor of Neurological Surgery, University of Illinois. 7½ × 5¼ in. Pp. 640, with 114 illustrations. 1960. Chicago: Year Book Publishers, Inc. (London: Interscience Publishers.) 68s.

THE three distinguished editors who produced the various parts of the previous year's review are responsible for the present one, a rather larger and somewhat more costly book. An introduction to the section of neurology is an interesting account of developments in the subject over the past ten years. While knowledge of the structure and origin of myelin, of the functions of oligodendroglia and the importance of the reticular formation of the brain has increased, electroencephalography has in some respects proved disappointing, especially when considered in regard to its clinical correlation. The practice of temporal lobectomy for psychomotor seizures has almost ceased and justly so, says the editor, who also points out that stellate ganglion block in cerebral thrombosis has died a natural death. The value of thymectomy in myasthenia gravis continues to be doubtful as does the part played by the thymus in causing it. In psychiatry the tranquillizers now hold second or third place as the largest group of drugs either prescribed or dispensed. Intervertebral disk lesions are considered at some length in the section on neurosurgery and it is noticed that spinal fusion after operations for these lesions is being less often performed. An interesting innovation in the surgery of the spinal cord is division of the tense dentate ligaments found in cases of subpial arterial vascular anomaly. It is suggested that intramedullary tension may be a cause of neurological deficit in these cases in which decompression alone has been disappointing and that releasing this tension by division of the ligaments may bring about improvement. Stereotaxic procedures again receive notice. Some satisfying results are reported, but there is a warning that personality changes may result from bilateral lesions in the thalamus and that complete and permanent abolition of rest tremor can be achieved only at the expense of a change in personality or in lasting disturbance of voluntary movements. Stereotaxic operations on the globus pallidus may become the procedure of choice for spasmodic torticollis which is so resistant to other methods of treatment. It is possible to refer to only a few of the contributions to which reference is made in this compact account of recent knowledge of the central nervous system, but it should prove as popular as ever with all who are interested in the subject.

The Sturge-Weber Syndrome. By G. L. ALEXANDER, M.D. (Edin.), F.R.C.S.E., F.R.C.S., Surgeon-in-Charge, Department of Neurological Surgery, Frenchay Hospital, Bristol; and R. M. NORMAN, M.D., M.R.C.P., Research Consultant in Neuropathology, The South Western Regional Hospital Board. 9½ × 6¾ in. Pp. 104 + viii, with 54 illustrations. 1960. Bristol: John Wright & Sons Ltd. 32s. 6d.

THIS is an excellent monograph devoted to a comprehensive survey of a relatively rare syndrome.

Although Sturge-Weber Syndrome may be said to have been born in 1879 when Sturge, a native of Bristol, first described the condition before the Clinical Society of London, yet Parkes Weber has written about this condition in medical journals since 1922. The authors of this monograph have dedicated it to Dr. Frederick Parkes Weber, now in his ninety-seventh year.

The Sturge-Weber syndrome is characterized clinically by cutaneous angiomatosis affecting the face, combined with epilepsy and often with gyriform calcifications. In a certain proportion of cases there is buphthalmos or glaucoma. Hemiparesis of the side of the body opposite to the facial nævus is often present. Homonymous hemianopia is also a feature of some of the patients.

The authors record complete clinical histories of five cases in which lobectomies have been carried out. These clinical details are well worth recording and there are some excellent photographs with them.

There is an admirable comprehensive list of references which covers eight pages.

As a monograph this book could not be bettered for it gives everything that is known about this syndrome; it is an excellent reference volume.

A Synopsis of Biology. By W. B. CROW, M.Sc., Ph.D., D.Sc., Senior Lecturer in Biology, Leicester College of Technology and Commerce. 7½ × 4¼ in. Pp. 1056 + xvi, with 327 illustrations. 1960. Bristol: John Wright & Sons Ltd. 90s.

THE author has covered his subject in a masterly way and although the text is brief, yet it is well chosen and very descriptive.

Wright's *Synopsis Series* is world famous, and this last addition to the series is well up to standard. This book should prove of real benefit to science students, both at home and abroad.

Fractures, Dislocations and Sprains. By PHILIP WILES, M.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S. 9½ × 12 in. Pp. 76, with 519 illustrations. 1960. London: J. & A. Churchill Ltd. 27s. 6d.

THIS book might be called an atlas of fractures and dislocations, for it contains a series of line drawings illustrating trauma of the skeleton. By the side of these drawings is placed a series of excellent radiographs

depicting every kind of fracture and dislocation. In the text, which is reduced to a minimum, a brief description is given of the various fractures. It should also be of use to medical officers who have to lecture on first aid.

Clinical Applications of Diagnostic and Therapeutic Nerve Blocks. By JOHN J. BONICA, M.D., Director, Department of Anaesthesiology, Tacoma General Hospital and Mountain View General Hospital. 8 × 5¼ in. Pp. 354, with 45 illustrations. 1960. Oxford: Blackwell Scientific Publications. 70s.

THIS is one of the American lecture series on Anaesthesiology [*sic*] edited by Dr. John Adriani, Director of the Department of Anaesthesia of the Charity Hospital in New Orleans. The title well describes the scope of the book which, beginning with a history of injections generally, goes on to a description of the agents used and the methods of their employment to produce analgesia, relieve pain, or promote physiological effects such as vasodilatation or suppression of reflexes. The opening chapter dealing with the history of the subject is rather sketchy, no mention being made of Carl Schleich's discovery of local analgesia and the uproar which his paper evoked at the meeting of the German Surgical Congress when it was reported. It is surprising to find Halsted's name misspelt in an American monograph though the mistake is frequently made in this country. The text is well interspersed with line drawings, reproductions of X-ray films, and other illustrations. There is a list of 150 references, and an index concludes this useful little concise and well-arranged guide to the subject.

Neoplasms of Bone and Related Conditions. By BRADLEY L. COLEY. M.D., Attending Surgeon (Emeritus), Bone Tumor Department, Memorial Hospital for Cancer and Allied Diseases. New York. Second edition. 10 × 6¾ in. Pp. 863, with 649 illustrations and 31 tables. 1960. New York: Paul B. Hoeber. $30.00.

THE second edition of this well-known work has been revised and extensively rewritten. New entities have been included and these include sections dealing with chondromyxoid fibroma, aneurysmal bone cyst, juxtacortical low-grade osteosarcoma, and malignant angioblastoma. There are quite a number of new illustrations including radiographs, clinical photographs, pathological specimens, and photomicrographs.

With the enormous material at the Memorial Sloan-Kettering Cancer Centre at his command, Dr. Coley has been able to write a very readable book on bone tumours. Much emphasis is placed on the role of radiation therapy, not only as a palliative agent or for inoperable tumours, but under certain conditions even as a preferred method of treatment.

An excellent reference volume.

Principles of Orthopaedic Surgery. By PAUL C. COLONNA, M.D., Professor Emeritus of Orthopædic Surgery, University of Pennsylvania Medical School. Revised edition. 9¼ × 6 in. Pp. 799, with 500 illustrations, 10 in colour. 1960. London: J. & A. Churchill Ltd. 150s.

THIS book is in reality a further development of the author's book on *Regional Orthopaedic Surgery*, which was published in 1950. It may be said to set forth the views and teaching of Dr. Paul Colonna, and these considerations are the outcome of a long life devoted to orthopædic surgery. The book is written in dogmatic yet lucid style, and is well illustrated, there being some 500 figures, ten of which are in colour. Quite a number of the fine illustrations are original.

The whole field of orthopædic surgery is covered in this book, and those portions which are devoted to fractures and dislocations are outstanding.

In this age of trauma students and practitioners alike will find this book full of useful information. As a reference book it could not be bettered.

A Practice of General Anæsthesia for Neurosurgery. By ROBERT I. W. BALLANTINE, M.R.C.S., L.R.C.P., D.A., F.F.A.R.C.S., Consultant Anæsthetist, St. Bartholomew's Hospital; and IAN JACKSON, M.R.C.S., L.R.C.P., D.A., Consultant Anæsthetist, St. Bartholomew's Hospital. 8 × 5¼ in. Pp. 152 + viii, with 68 illustrations. 1960. London: J. & A. Churchill Ltd. 27s. 6d.

THIS is a well-written and useful little book. It deals concisely but adequately with most of the problems which face the anæsthetist in neurosurgical operations. It is obvious that the authors are thoroughly at home in this field, and their recommendations are sane and practical. The well-kept anæsthesia records with which the text is interspersed illustrate the points made most effectively. The book can be recommended both for the occasional and for the regular neurosurgical anæsthetist.

A Clinical Prospect of the Cancer Problem. By D. W. SMITHERS, M.D., F.R.C.P., F.F.R. 9¾ × 6¼ in. Pp. 232 + xvi, with 46 illustrations. 1960. Edinburgh & London: E. & S. Livingstone Ltd. 37s. 6d.

THIS is the introductory volume to a series of monographs dealing with neoplastic disease at various sites. Professor Smithers is the general editor of these monographs.

In the present volume Professor Smithers covers the important field of cancer in a very broad way. Every facet on the cancer problem is covered, and the author's extensive knowledge of the literature is quite manifest in his criticism of other people's work. He gives an interesting and clear account of the theories of causation of cancer. The question of hormones and the part they play in cancer production are well described.

There is one of the most comprehensive authors' indexes yet published, and there is a subject index as well.

After reading this monograph, one cannot help feeling that the author takes a very pessimistic view as far as the cure of cancer is concerned. He does not appear to hold any hope of a cure of this disease at all. In spite of this view, the author has written a stimulating book.

Typical Gynecologic Operations. By DR. SIEGFRIED TAPFER, in charge of the University Clinic for Gynecology and Obstetrics, Innsbruck, Tirol, Austria. Translated by L. M. SZAMEK, M.D. 9¾ × 7 in. Pp. 81, with 167 illustrations. 1960. Philadelphia: J. B. Lippincott Co. (Pitman Medical Publishing Co. Ltd). 72s.

THIS is more or less an atlas of gynæcological operations depicting the various procedures as carried out in Dr. Tapfer's Clinic in Innsbruck. The illustrations are excellent and are by Dr. Walter Wenzel, an artist of distinction, and, being a medical man with a full understanding of the procedures he depicts, he has portrayed graphically the step-by-step operative techniques involved. Vaginal hysterectomy is perhaps the best illustrated operation in the book.

A useful book for the student and resident medical officer.

Cosmetic Surgery. Principles and Practice. By SAMUEL FOMAN, M.D., Director of Plastic Surgery, Manhattan General Hospital, New York. 10 × 7 in. Pp. 651, with 608 illustrations. 1960. Philadelphia: J. B. Lippincott Co. (Pitman Medical Publishing Co. Ltd.) £11.

THE two World Wars have made plastic surgery what it is to-day; and the ever increasing road accidents have also brought to the fore the great importance of this branch of surgery.

This book is a very comprehensive account of plastic surgery as practised in the United States to-day. This book covers in a very practical way the entire subject and is lavishly illustrated. It is organized in two main sections. The first, General Principles, covers general pre- and post-operative considerations; anæsthesia; incision and closure; wounds; scars; and tissue transplantation. The second, Regional Cosmetic Surgery, covers cosmetic operations on the ear and eye, and maxillofacial surgery.

This is a noteworthy text-book, and one that will be popular with practitioners and students.

The Early History of Surgery. By W. J. BISHOP. 8½ × 5½ in. Pp. 192. Illustrated. 1960. London: Robert Hale Ltd. 18s.

THIS is a most useful and interesting book, and we expected that it would be, for few people know more of the history of Surgery than the author. Mr. Bishop has graduated and matured in Medical Libraries and therefore is steeped in medical literature.

It is no easy matter to sift the enormous material which has accumulated dealing with the early history of medicine, and then write a book which will appeal both to the medical profession and the laity. Yet this is what Mr. Bishop has accomplished with great credit. He has managed to portray the great surgeons of the past in a fascinating way.

Practitioners, students, and nurses will find this an intriguing and attractive book, which contains a wealth of material of interest to them.

Intra-osseous Venography. By ROBERT A. SCHOBINGER, M.D., New York. 6½ × 10 in. Pp. 243, with 317 illustrations. 1960. New York: Grune & Stratton Inc. $14.50.

THIS small monograph, with over three hundred radiographs and figures depicting normal and abnormal venous patterns, is quite unique. The author uses the term 'osteomyolography', and injects sodium diatrizoate as the contrast medium. The injection is given into the bone at sites of election which are listed. There are certain contra-indications to this procedure such as asthma, urticaria, allergy, and idiosyncrasy to the contrast medium.

Osteomyelography can be used in the detection of hidden primary or metastatic bone tumours and other bone conditions.

This book should prove of value to all those who wish to use this method of investigation.

Surgical Anatomy of the Bronchovascular Segments. By WILLIAM E. BLOOMER, M.D., Assistant Professor of Surgery, Yale University School of Medicine; AVERILL A. LIELOW, M.D., John Slade Ely Professor of Pathology, Yale University School of Medicine; and MILTON R. HALES, M.D., Assistant Professor of Pathology, Yale University School of Medicine. 11 × 8½ in. Pp. 273, with 208 illustrations. 1960. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) £6 12s.

THE precise anatomical arrangements of the bronchi and their numerous branches, together with the intimate relationships of the vascular structures at the hilum of the lung, are of the greatest importance to the thoracic surgeon. This being so it is essential that books like this and that of Sir Russell Brock should be available for quick reference.

The modern development of plastics has allowed anatomists and pathologists to outline the finer bronchi, and reveal the relationships of these with the small vascular tributaries. The time has passed when mass ligature of the base of the lung was performed. To-day, partial lobectomy is possible on a very firm footing. Although pulmonary tuberculosis does not demand much surgery because of the antibiotics, yet cancer of the lung is increasing year by year, and so it is very necessary for the thoracic surgeon to have a detailed knowledge of the anatomical components of the bronchial tree.

This book, which in reality is an atlas, is completely up to date and will take its place as a most valuable reference volume.

The Liver and Cancer. A New Cancer Theory. By KASPER BLOND, M.D., F.I.C.S. Second edition. 8½ × 5½ in. Pp. 400 + xvi. 1960. Bristol: John Wright & Sons Ltd. 35s.

THE second edition of this book has been revised, and some 170 additional pages have been incorporated.

The author, who has studied the cancer problem for many years, tries to prove that cancer is the end-result of a chronic nutritional disorder. Dr. Blond certainly marshals his facts, and is convincing in his evidence from the literature.

This book should be read by all who have to treat cancer, for it opens up a new conception which may well help eventually in the cure of this disease. Anyone who who can throw new light on this disease should be allowed to state his case.

Current Surgical Management, II. Edited by JOHN H. MULHOLLAND, M.D., Editor-in-Chief; EDWIN H. ELLISON, M.D.; STANLEY R. FRIESER, M.D., with contributions by 50 authorities. 9¾ × 6¾. Pp. 348. Illustrated. 1960. Philadelphia and London: W. B. Saunders Co. 56s.

THIS is the second volume on *Current Surgical Management* and it contains the thoughts and teaching of some fifty American authorities. Each contributor writes on a subject which he has made his own.

It is impossible to review this book in detail as the articles range over the whole field of surgery from carcinoma of the breast to the pathogenesis and management of pilonidal sinus. A large proportion of the book is devoted to the treatment of gastro-intestinal conditions, and this section without doubt contains the best sections. The illustrations are good and have been well chosen, especially those which depict operative technique.

This book should prove of real value to surgical registrars who are contemplating higher surgical degrees or diplomas.

Essays on the First Hundred Years of Anæsthesia. Volume I. By W. STANLEY SYKES, M.B.E., M.B., B.Chir. (Cantab.), D.A. 8¾ × 6¼ in. Pp. 171, with 19 figures and 39 plates. 1960. Edinburgh and London: E. & S. Livingstone Ltd. 30s.

THIS book gives a readable and digressive account of the early days of anæsthesia and can be read with enjoyment by anyone to whom this is not already familiar ground.

Dr. Sykes has a ready pen and an inquiring mind which leads up some bypaths far away from the title of his book. This is not a disadvantage, for most of those who already have some knowledge of the history of anæsthesia find the reiteration of the stories of Hickman and Davey, of Morton and Wells, of Simpson and Syme, distinctly tedious.

The digressions by which this tedium is lightened lead to such surprises as the training of generals and the wages of "trouser finishers". A pleasant chapter is devoted to curare, in which there is some new material, but we are not spared a repetition of the hackneyed story of the she-ass.

Though there are many light and pleasant touches in the book, it is difficult to find much justification for its appearance. The author's original object, we are told, was to make a comprehensive encyclopædia of every type of early apparatus connected with anæsthesia, but he soon found this to be an impossible task, and has served up instead a volume—evidently intended as the first of

several—which certainly includes some pictures and descriptions of early devices, but which consists mainly of familiar stuff.

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Volume 2: This comprehensive volume contains coloured plates showing both male and female reproductive anatomy, physiology, and pathology. Dr. Netter, the artist, has given of his best in portraying the pathology of both primary and secondary male and female sex organs.

As a reference volume this could not be bettered, and both students and practitioners will find it most useful.

Volume 3: The first part of Volume 3 is devoted to the upper digestive tract and consists of some 172 coloured plates of the anatomy of the mouth and pharynx, œsophagus, stomach, and duodenum. Following these are a series of paintings illustrating the various pathological conditions which may arise in these organs. The coloured plates are pre-eminent and are some of the best we have ever seen.

Volume 3, *Part III*: This third part comprises some 133 coloured plates depicting the liver, biliary tract, and pancreas in health and disease. The pictures have been well chosen and all have been beautifully executed.

These four volumes of coloured plates make up a reference library in themselves. There is also a very extensive bibliography, which is always useful. Ciba have indeed put the medical and nursing professions greatly in their debt by the publication of these excellent coloured atlases, and, what is more, selling them to the profession at cost price.

Bibliotheca Gastroenterologica, Fasc. 2. **Dünndarmpathologie.** Pathologie de l'intestin grêle. Symposium der Schweizerischer Gesellschaft für Gastroenterologie in Genf. 1959. By H. J. FAHRLANDER. 9¾ × 6¾ in. Pp. 220, with 54 illustrations. 1960. Basel and New York: S. Karger. S.Fr.38.

A NOTICEABLE feature of post-war gastro-enterology has been the surge of new interest taken in the subject of the small intestine. New techniques of radiological, histological, and biochemical investigation have been introduced, new disease entities described, and many new facts discovered about those well recognized but still mysterious conditions, the steatorrhœas and regional ileitis.

The material in this Swiss symposium on the pathology of the small intestine is wider in its scope than the title suggests. Sixteen papers (ten in French and six in German) are presented, dealing with physiology, diagnostic procedures, morphology, radiology, clinical problems, and surgery.

A short but useful summary in English is given after each paper.

Shaw's Textbook of Operative Gynæcology. Revised by JOHN HOWKINS, M.D., M.S. (Lond.), F.R.C.S. (Eng.), F.R.C.O.G., Obstetrical and Gynaecological Surgeon, St. Bartholomew's Hospital, London. Second edition. 11 × 8½ in. Pp. 484 + viii, with 428 illustrations. 1960. Edinburgh and London: E. & S. Livingstone Ltd. £5 5s.

THE second edition of this capacious text-book is the result of the able editorship of John Howkins, who has done an excellent job.

When the first edition appeared in 1954 everyone agreed that it was Shaw's masterpiece, and the best text-book of operative gynæcology in the English language.

The new edition is certainly a worthy successor to the first one, written by the original author.

Throughout this large volume the illustrations, over 400 in number, are admirable, especially the coloured ones.

There can be little doubt that this book will continue to be a reference volume to all those surgeons who undertake gynæcological operations.

Fundamental Aspects of Normal and Malignant Growth. Edited by WIKTOR W. NOWINSKI. 9¾ × 6½ in. Pp. 1025 + xvi, illustrated. 1960. Amsterdam: Elsevier Publishing Co. (London: Van Nostrand Co. Ltd.) £10 10s.

THIS is a most interesting book covering all aspects of growth from the simple organisms to the complex higher animals.

It is written by a select body of contributors who cover all fields, including the problems of basic research in cancer. The book gives an excellent survey of such phenomena as organization and induction, growth in tissue culture, regeneration of growth, and metabolism of the cancer cell.

A very stimulating chapter is devoted to wound healing as a problem of growth. The questions of wound closure and epithelialization, granulation tissue and its chemistry, are expounded in a very able manner.

The measurement of growth and cell division and enlargement makes fascinating reading.

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There is a full and comprehensive list of references at the end of each chapter.

Dr. Nowinski has certainly produced a very exciting volume, full of important facts and numerous excellent illustrations.

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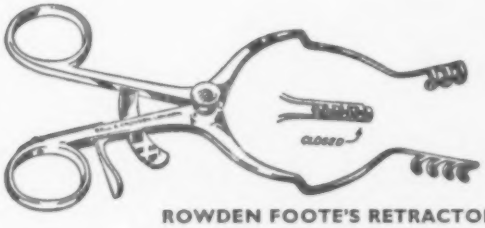

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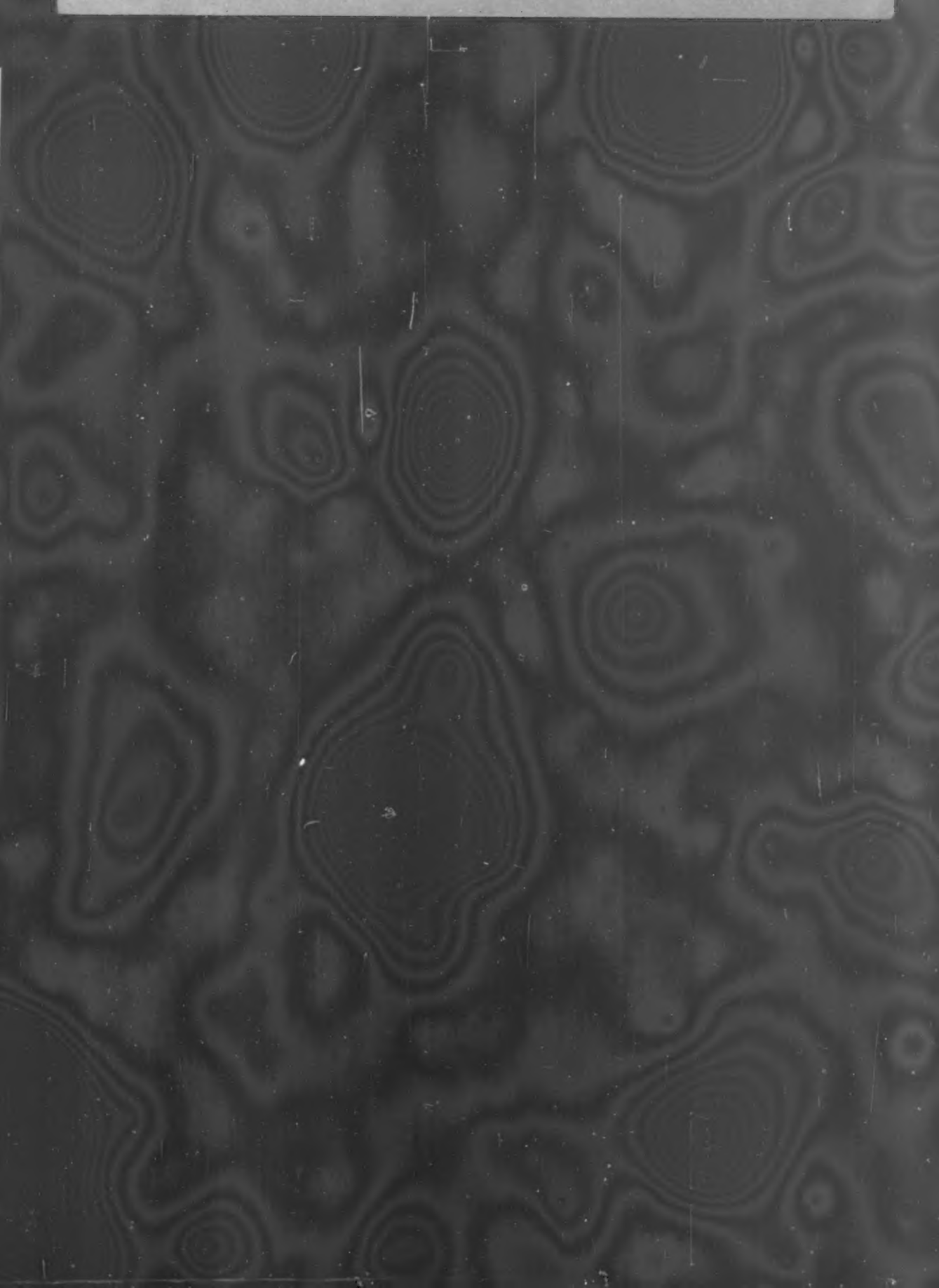
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